

Connecting via Winsock to Dialog

Logging in to Dialog

Trying 31060000009998...Open

DIALOG INFORMATION SERVICES

PLEASE LOGON:

ENTER PASSWORD:

Welcome to DIALOG

Dialog level 05.24.00D

Last logoff: 28may09 09:23:05

Logon file405 02jun09 07:11:02

*** ANNOUNCEMENTS ***

*** FREE FILE OF THE MONTH (JUNE)

Inspec (File 2)

Derwent World Patents Index First View Overview (File 331)

Each month Dialog offers an opportunity to try out new or unfamiliar sources by offering \$100 of free searching (either DialUnits or connect time) in one specific file. Output and Alerts charges are not included. For more details visit: <http://www.dialog.com/freefile/> and then take a moment to get familiar with another great Dialog resource.

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NEW FILE

***File 457, The Lancet(R)

RESUMED UPDATING

***File 523, D&B European Financial Records

RELOADS COMPLETED

***File 658, TRADEMARKSCAN(R) - Benelux

***File 659, TRADEMARKSCAN(R) - Denmark
***File 661, TRADEMARKSCAN(R) - Switzerland
***File 662, TRADEMARKSCAN(R) - Austria
***File 669, TRADEMARKSCAN(R) - Japan
***File 678, TRADEMARKSCAN(R) - Norway

FILES REMOVED

***File 301, CHEMNAME - please use File 398 ChemSearch
***File 388, PEDS: Defense Program Summaries
***File 588, DMS-FI Contract Awards

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>>>and events, please visit What's New from Dialog at <<<
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>>>a specific database by entering HELP NEWS <file number>. <<<

* * *

SYSTEM:HOME

Cost is in DialUnits

Menu System II: D2 version 1.8.0 term=ASCII

*** DIALOG HOMEBASE(SM) Main Menu ***

Information:

1. Announcements (new files, reloads, etc.)
2. Database, Rates, & Command Descriptions
3. Help in Choosing Databases for Your Topic
4. Customer Services (telephone assistance, training, seminars, etc.)
5. Product Descriptions

Connections:

6. DIALOG(R) Document Delivery
7. Data Star(R)

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/H = Help

/L = Logoff

/NOMENU = Command Mode

Enter an option number to view information or to connect to an online service. Enter a BEGIN command plus a file number to search a database

(e.g., B1 for ERIC).

? b 410

02jun09 07:11:03 User226352 Session D1140.1

\$0.00 0.275 DialUnits FileHomeBase

\$0.00 Estimated cost FileHomeBase

\$0.00 Estimated cost this search

\$0.00 Estimated total session cost 0.275 DialUnits

File 410:Dialog Customer Newsletters 2008

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Set	Items	Description
---	-----	-----

? set hi ;set hi

HIGHLIGHT set on as ''

HIGHLIGHT set on as ''

? b biochem

02jun09 07:11:06 User226352 Session D1140.2		
\$0.00	0.117	DialUnits File410
\$0.00		Estimated cost File410
\$0.02		TELNET
\$0.02		Estimated cost this search
\$0.02		Estimated total session cost 0.392 DialUnits

SYSTEM:OS - DIALOG OneSearch

File 5:Biosis Previews(R) 1926-2009/May W4
(c) 2009 The Thomson Corporation

File 6:NTIS 1964-2009/May W4
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File 24:CSA Life Sciences Abstracts 1966-2009/Jul
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File 40:Enviroline(R) 1975-2008/May
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*File 40: This file is closed and will no longer update. For similar data, please search File 76-Environmental Sciences.

File 41:Pollution Abstracts 1966-2009/Jul
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File 65:Inside Conferences 1993-2009/Jun 01
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File 73:EMBASE 1974-2009/May 29
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File 76:Environmental Sciences 1966-2009/Jul
(c) 2009 CSA.

File 98:General Sci Abs 1984-2009/Jun
(c) 2009 The HW Wilson Co.

File 103:Energy SciTec 1974-2009/May B1
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*File 103: For access restrictions see Help Restrict.

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(c) 2007 CSA.

*File 136: This file is closed.

File 143:Biol. & Agric. Index 1983-2009/May
(c) 2009 The HW Wilson Co

File 144:Pascal 1973-2009/May W5
(c) 2009 INIST/CNRS

File 154:MEDLINE(R) 1990-2009/May 29
(c) format only 2009 Dialog

File 155:MEDLINE(R) 1950-2009/May 29
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File 156:ToxFile 1965-2009/May W4
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File 162:Global Health 1983-2009/May W4
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*File 162: The file has been reloaded and accession numbers have
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File 172:EMBASE Alert 2009/May 29
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File 305:Analytical Abstracts 1980-2009/Apr W3
(c) 2009 Royal Soc Chemistry

*File 305: Alert feature enhanced for multiple files, duplicate
removal, customized scheduling. See HELP ALERT.

File 369:New Scientist 1994-2009/May W4
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File 370:Science 1996-1999/Jul W3
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*File 370: This file is closed (no updates). Use File 47 for more
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File 393:Beilstein Database - Abstracts 2008/Q2
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File 399:CA SEARCH(R) 1967-2009/UD=15023
(c) 2009 American Chemical Society

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IPCR/8 classification codes now searchable as IC=. See HELP NEWSIPCR.

File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
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Set	Items	Description
---	-----	-----
? s adiponectin		
	S1 49381	ADIPONECTIN
? s s1 and (multimer or aggregat?)		
	49381	S1
	9927	MULTIMER
	1297324	AGGREGAT?
	S2 464	S1 AND (MULTIMER OR AGGREGAT?)
? rd s2		

>>>Duplicate detection is not supported for File 393.

>>>Records from unsupported files will be retained in the RD set.
S3 110 RD S2 (unique items)
? t s3/7/1=10
>>>'=' not allowed in command
? t s3/7/1-10
>>>Format 7 is not valid in file 143

3/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2009 The Thomson Corporation. All rts. reserv.

0020883139 BIOSIS NO.: 200900223473
Comparison of Immunoassays for the Selective Measurement of Human
High-Molecular Weight Adiponectin
AUTHOR: Liu Dan; Schuster Tibor; Baumann Marcus; Roos Marcel;
Sollinger
Daniel; Lutz Jens; Heemann Uwe; von Eynatten Maximilian (Reprint)
AUTHOR ADDRESS: Tech Univ Munich, Dept Nephrol, Ismaningerstr 22,
D-81675
Munich, Germany**Germany
AUTHOR E-MAIL ADDRESS: maximilian.eynatten@lrz.tum.de
JOURNAL: Clinical Chemistry 55 (3): p568-572 MAR 2009 2009
ISSN: 0009-9147
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: BACKGROUND: Adiponectin is an adipocyte-derived hormone circulating in different multimer complexes. The high-molecular-weight (HMW) complex is likely the active form of this protein and has been recognized as a risk marker for type 2 diabetes and coronary artery disease (CAD). Because quantification of HMW adiponectin by Western blot analysis is time-consuming, novel ELISAs have been developed to simplify measurements in clinical research. However, these enzyme immunoassays have not been cross-validated in larger patient groups. We evaluated 2 individual ELISA systems by comparison to Western blotting for measurement of the distribution of HMW adiponectin in healthy individuals and patients with CAD and type 2 diabetes.METHODS: We measured HMW adiponectin in 204 individuals (83 CAD patients, 81 type 2 diabetes patients, and 40 healthy controls). Correlations, range of agreement, and imprecision of HMW concentrations obtained using 2 commercial ELISAs (#1, ALPCO Diagnostics; #2, Millipore) were evaluated by comparison with quantitative Western blotting.RESULT:

Adiponectin results of the ELISAs were significantly correlated with those obtained by Western blotting (both $r > 0.75$, $P < 0.001$). Deming regression and Bland-Altman analyses indicated high agreement among the 3 immunoassays. The median difference between HMW adiponectin concentrations measured by ELISA and by Western blot was +0.4 mg/L for ELISA #1 and -0.4 mg/L for ELISA #2 with 95% of value

differences < 3 mg/L. CONCLUSIONS: Selective measurement of HMW adiponectin by ELISA is feasible; however, individual differences among immunoassays must be considered. The evaluated ELISAs exhibit analytical characteristics that allow their use as equivalent for Western blot analysis in larger clinical and epidemiological groups.

3/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0020786289 BIOSIS NO.: 200900126623
Fish oil, but not flaxseed oil, decreases inflammation and prevents pressure overload-induced cardiac dysfunction
AUTHOR: Duda Monika K; O'Shea Karen M; Tintinu Anselm; Xu Wenhong; Khairallah Ramzi J; Barrows Brian R; Chess David J; Azimzadeh Agnes M; Harris William S; Sharov Victor G; Sabbah Hani N; Stanley William C (Reprint)
AUTHOR ADDRESS: Univ Maryland, Dept Med, Div Cardiol, 20 Penn St, HSF2, Room S022, Baltimore, MD 21201 USA**USA
AUTHOR E-MAIL ADDRESS: wstanley@medicine.umaryland.edu
JOURNAL: Cardiovascular Research 81 (2): p319-327 FEB 1 2009 2009
ITEM IDENTIFIER: doi:10.1093/cvr/cvn310
ISSN: 0008-6363
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Clinical studies suggest that intake of omega-3 polyunsaturated fatty acids (omega-3 PUFA) may lower the incidence of heart failure. Dietary supplementation with omega-3 PUFA exerts metabolic and anti-inflammatory effects that could prevent left ventricle (LV) pathology; however, it is unclear whether these effects occur at clinically relevant doses and whether there are differences between omega-3 PUFA from fish [eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)] and vegetable sources [alpha-linolenic acid (ALA)]. We assessed the development of LV remodelling and pathology in rats subjected to aortic banding treated with omega-3 PUFA over a dose range that spanned the intake of humans taking omega-3 PUFA supplements. Rats

were fed a standard food or diets supplemented with EPA+DHA or ALA at 0.7, 2.3, or 7% of energy intake. Without supplementation, aortic banding increased LV mass and end-systolic and -diastolic volumes. ALA supplementation had little effect on LV remodelling and dysfunction. In contrast, EPA+DHA dose-dependently increased EPA and DHA, decreased arachidonic acid in cardiac membrane phospholipids, and prevented the increase in LV end-diastolic and -systolic volumes. EPA+DHA resulted in a dose-dependent increase in the anti-inflammatory adipokine adiponectin, and there was a strong correlation between the prevention of LV chamber enlargement and plasma levels of adiponectin ($r = -0.78$). Supplementation with EPA+DHA had anti-aggregatory and anti-inflammatory effects as evidenced by decreases in urinary thromboxane B-2 and serum tumour necrosis factor- α . Dietary supplementation with omega-3 PUFA derived from fish, but not from vegetable sources, increased plasma adiponectin, suppressed inflammation, and prevented cardiac remodelling and dysfunction under pressure overload conditions.

3/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0020723112 BIOSIS NO.: 200900063446
Differential effects of bariatric surgery-induced weight loss on adiponectin multimer complexes
AUTHOR: Linscheid Philippe (Reprint); Christ-Crain Mirjam; Stoeckli Rolf;
Mueller Beat; Keller Ulrich
AUTHOR ADDRESS: Univ Basel Hosp, Dept Res, CH-4031 Basel, Switzerland**
Switzerland
JOURNAL: International Journal of Obesity 32 (Suppl. 6): pS77 DEC 2008
2008
CONFERENCE/MEETING: 4th Fribourg Obesity Research Conference Fribourg, SWITZERLAND 20070914,
ISSN: 0307-0565
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

3/7/4 (Item 4 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)
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0020720253 BIOSIS NO.: 200900060587

High molecular weight adiponectin correlates positively with
myeloperoxidase in patients with type 2 diabetes mellitus

AUTHOR: Bobbert P (Reprint); Rauch U; Stratmann B; Goldin-Lang P;
Antoniak

S; Bobbert T; Schultheiss H P; Tschoepe D

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AUTHOR E-MAIL ADDRESS: peter.bobbert@charite.de

JOURNAL: Diabetes Research and Clinical Practice 82 (2): p179-184
NOV 2008

2008

ITEM IDENTIFIER: doi:10.1016/j.diabres.2008.07.018

ISSN: 0168-8227

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Adiponectin (APN) is present in human plasma as a low
molecular weight (LMW), a middle molecular weight (MMW) and a high
molecular weight form (HMW). As a support to determine properties
such as

anti-atherogenic or atherogenic effects, recent clinical studies
suppose

to deter-mine the ratio of each APN multimer to total APN but not
the absolute plasma concentration of APN. In the present study, the
correlation of APN and its multimers with myeloperoxidase (MPO), an
enzyme with pro-inflammatory proper-ties, was examined in patients
with

type 2 diabetes mellitus. MPO and APN serum levels were assessed in
49

patients with type 2 diabetes mellitus at the beginning and at the
end of

an anti-diabetic treatment. After treatment a significant increase
in the

ratio of HMW to total APN (from 0.43 +/- 0.16 to 0.59 +/- 0.14, $p < 0.05$)

was found. Before treatment, HMW-APN was correlated positively with
MPO

($r = 0.314$, $p < 0.05$). Moreover, a positive correlation was observed
between the increased HMW ratio and MPO during treatment ($r =$
0.304, $p <$

0.05). HMW-APN correlates positively with MPO in patients with type 2
diabetes. Therefore, HMW-APN may exert possible pro-inflammatory
effects

in type 2 diabetes. (C) 2008 Elsevier Ireland Ltd. All rights
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3/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0020587007 BIOSIS NO.: 200800633946
Adiponectin multimer distribution in patients with familial
combined hyperlipidemia
AUTHOR: Koenen Tim B (Reprint); van Tits Lambertus J H; Holewijn
Suzanne;
Lemmers Heidi L M; den Heijer Martin; Stalenhoef Anton F H; de Graaf
Jacqueline
AUTHOR ADDRESS: Radboud Univ Nijmegen, Med Ctr, Dept Gen Internal Med
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POB 9101, NL-6500 HB Nijmegen, Netherlands**Netherlands
AUTHOR E-MAIL ADDRESS: T.Koenen@aig.umcn.nl
JOURNAL: Biochemical and Biophysical Research Communications 376
(1): p
164-168 NOV 7 2008 2008
ITEM IDENTIFIER: doi:10.1016/j.bbrc.2008.08.111
ISSN: 0006-291X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin is secreted from adipocytes in different
multimers, of which the high molecular weight (HMW) form is
supposed to
mediate favorable metabolic and anti-atherogenic effects. We
determined
adiponectin multimers in 29 female and 22 male patients with
familial combined hyperlipidemia (FCH) and 51 age-, gender-, and
BMI-matched controls in relation to cardiovascular disease (CVD). We
observed a clear sexual dimorphism of total adiponectin and its
multimers. Female, but not male, FCH patients had significant lower
total
adiponectin and both HMW and low molecular weight (LMW)
adiponectin than controls. The adiponectin sensitivity index
(ASI), reflected by HMW/total adiponectin, and the LMW/HMW
adiponectin ratio did not differ significantly between FCH females
and control females. However, FCH females with CVD exhibited
significantly lower ASI (34.2 +/- 10.1% vs 46.0 +/- 7.1%) and higher
LMW/HMW ratio (1.5 +/- 0.8 vs 0.7 +/- 0.3) compared to FCH females
without CVD, reflecting a more atherogenic adiponectin
multimer distribution. (C) 2008 Elsevier Inc. All rights reserved.

3/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0020549023 BIOSIS NO.: 200800595962

Familial aggregation in patients with non-alcoholic steatohepatitis
AUTHOR: Tokushige Katsutoshi (Reprint); Yatsuji Satoru; Hashimoto
Etsuko;

Kabutake Ayae; Tobari Maki; Taniai Makiko; Shiratori Keiko
AUTHOR ADDRESS: Tokyo Womens Med Univ, Dept Med and Gastroenterol,
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AUTHOR E-MAIL ADDRESS: ktoku@pg7.so-net.ne.jp

JOURNAL: Internal Medicine (Tokyo) 47 (5): p405-410 2008 2008

ITEM IDENTIFIER: doi:10.2169/internalmedicine.47.0476

ISSN: 0918-2918

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: We encountered three families that showed NASH
accumulation. In

family #1, a 21-year-old son and 10-year-old daughter were
diagnosed with

nonalcoholic steatohepatitis (NASH). They shared two adiponectingene
single nucleotide polymorphisms (SNP). In family #2, a 51-year-old
mother

and 27-year-old son were diagnosed with NASH and shared the SNPs of
other

genes. In family #3, a 66-year-old mother and 34-year-old son were
diagnosed with NASH and shared the SNPs of other genes. SNP sites
differed among the three families, suggesting that the genes
associated

with the occurrence of NASH might be different in each patient.

3/7/7 (Item 7 from file: 5)
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0020485825 BIOSIS NO.: 200800532764

Adipocyte adhesion molecule (ACAM) inhibits adipocyte hypertrophy in
obesity

AUTHOR: Murakami Kazutoshi (Reprint); Wada Jun; Nakatsuka Atsuko;
Kanzaki

Motoko; Teshigawara Sanae; Terami Takahiro; Inoue Kentaro; Makino
Hirofumi

AUTHOR ADDRESS: Okayama, Japan**Japan

JOURNAL: Diabetes 57 (Suppl. 1): pA110-A111 JUN 2008 2008

CONFERENCE/MEETING: 68th Annual Meeting of the
American-Diabetes-Association San Francisco, CA, USA June 06 -10,
2008;

20080606

SPONSOR: Amer Diabet Assoc

ISSN: 0012-1797

DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Citation

LANGUAGE: English

3/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0020476411 BIOSIS NO.: 200800523350
The effects of phytosteryl ferulates on multimeric form of
adiponectin secreted from 3T3-L1 adipocytes
AUTHOR: Ohara K (Reprint); Nagasaka R; Ushio H
AUTHOR ADDRESS: Tokyo Univ Marine Sci and Technol, Tokyo, Japan**Japan
JOURNAL: FEBS Journal 275 (Suppl. 1): p140 JUN 2008 2008
CONFERENCE/MEETING: Joint Conference of the 33rd FEBS Congress/11th
IUBMB
Conference Athens, GREECE June 28 -July 03, 2008; 20080628
SPONSOR: Federat Biochem Soc
Int Union Biochem & Mole Biol
ISSN: 1742-464X_(print) 1742-4658_(electronic)
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

3/7/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0020393277 BIOSIS NO.: 200800440216
High molecular mass multimer complexes and vascular expression
contribute to high adiponectin in the fetus
AUTHOR: Pinar H; Basu S; Hotmire K; Laffineuse L; Presley L;
Carpenter M;
Catalano P M; Hauguel-de Mouzon S (Reprint)
AUTHOR ADDRESS: Case Western Reserve Univ, MetroHlth Med Ctr, Dept
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Biol, 2500 MetroHlth Dr, Cleveland, OH 44109 USA**USA
AUTHOR E-MAIL ADDRESS: shdemouzon@metrohealth.org
JOURNAL: Journal of Clinical Endocrinology & Metabolism 93 (7):
p2885-2890
JUL 1 2008 2008
ITEM IDENTIFIER: doi:10.1210/jc.2008-0009
ISSN: 0021-972X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Context: High plasma adiponectin concentrations in human
fetuses and neonates are unique features of early developmental
stages.

Yet, the origins of the high adiponectin concentrations in the
perinatal period remain elusive.Objective: This study was
undertaken to

identify the sources and functional properties of adiponectin in utero.Design and Methods: Tissue specimens were obtained at autopsy from

21- to 39-wk-old stillborn human fetuses. Adipose tissue and placenta

were obtained at term elective cesarean section. Adiponectin complexes and expression were measured by immunodetection and real-time

PCR.Results: Adiponectin mRNA transcripts were detected in fetal sc and omental adipose depots at lower concentrations than in maternal adipose tissue. Immunoreactive adiponectin was also observed in vascular endothelial cells of fetal organs, including skeletal muscle,

kidney, and brain. The absence of adiponectin in all placental cell types and lack of correlation between maternal and umbilical adiponectin indicate that umbilical adiponectin reflects its exclusive production by fetal tissues. The most prominent forms of adiponectin in fetal plasma were high and low molecular mass (HMW and LMW) multimers of 340 and 160 kDa, respectively. The proportion of

the HMW complexes was 5-fold ($P < 0.001$) higher in umbilical plasma than

in adult. The high HMW and total adiponectin levels were associated with lower insulin concentration and lower homeostasis model of assessment of insulin resistance indices in umbilical plasma, reflecting

higher insulin sensitivity of the fetus compared with adult.Conclusions:

The abundance of HMW adiponectin and its vascular expression are characteristics of human fetal adiponectin. Combined with high insulin sensitivity, fetal adiponectin may be a critical determinant of in utero growth.

3/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0020369310 BIOSIS NO.: 200800416249

Family history and familial aggregation in patients with nonalcoholic steatohepatitis

AUTHOR: Noto Haruka; Tokushige Katsutoshi; Hashimoto Etsuko; Kabutake Ayae;

Tobari Maki; Yatsuji Satoru; Taniai Makiko; Shiratori Keiko
JOURNAL: Gastroenterology 134 (4, Suppl. 1): pA784 APR 2008 2008
CONFERENCE/MEETING: Digestive Disease Week Meeting/109th Annual Meeting of

the American-Gastroenterological-Association San Diego, CA, USA May 17

-22, 2008; 20080517

SPONSOR: Amer Gastroenterol Assoc

ISSN: 0016-5085

DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Genetic background contributes to the onset / progress of nonalcoholic steatohepatitis (NASH), as with other lifestyle-related diseases. In order to investigate the role of genetic background in NASH,

we investigated the frequencies of diabetes mellitus (DM), hypertension

(HT), and hyperlipidemia (HL) in the families of NASH patients. In addition, we found 3 families with NASH aggregation and investigated their single nucleotide polymorphisms (SNP). (Method)

We examined 106 NASH patients and 37 control subjects. We compared the frequency of intrafamilial DM / HT/ HL by questionnaire. In three families with NASH aggregation, SNPs of TNF, adiponectin, beta 3-adrenergic receptor, interleukin- 1, MTP, MnSOD genes were examined. (Results)1. The frequency of DM in parents of NASH

patients (27%) is significantly higher than that of parents of controls (7%). A

trend toward DM was noted in the mothers, but not the fathers. The frequency of DM in brothers of NASH patients was also higher than that of

controls. Regarding HT and HL, there was no difference between two groups. 2. In 106 NASH patients, we found three families that showed NASH

accumulation. In family #1, a 21-year-old son and 10-year-old daughter

were diagnosed with NASH and shared two adiponectin gene SNPs. In family #2, a 51-year-old mother and 27-year-old son were diagnosed with

NASH and shared SNPs for TNF, beta 3-adrenergic receptor and MTP. In family #3, a 66-year-old mother and 34-year-old son were diagnosed with

NASH and shared SNPs for MTP and MnSOD. SNP sites reported to be associated with NASH or DM, differed among the three families. in all

patients, liver function was correlated with body weight. (Conclusion)

Genomic background linked to DM might be related to the pathogenesis of

NASH. However, critical SNP sites differed among the three families with

NASH aggregation, suggesting that the genes associated with its occurrence might be different in each patient. In addition, if a person's

genetic background is associated with the onset or progress of NASH, amelioration of living habits remains important.

? ds

Set	Items	Description
S1	49381	ADIPONECTIN
S2	464	S1 AND (MULTIMER OR AGGREGAT?)
S3	110	RD S2 (unique items)

? t s3/7/11-110

>>>Format 7 is not valid in file 143

3/7/11 (Item 11 from file: 5)
 DIALOG(R)File 5:Biosis Previews(R)
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0020367692 BIOSIS NO.: 200800414631

The association of circulating adiponectin multimers with Barrett's esophagus

AUTHOR: Rubenstein Joel H; Kao John Y; Madanick Ryan D; Zhang Min; Wang

Meizhi; Spacek Melissa; Donovan Jena; Bright Stephanie D; Shaheen Nicholas J

JOURNAL: Gastroenterology 134 (4, Suppl. 1): pA437 APR 2008 2008

CONFERENCE/MEETING: Digestive Disease Week Meeting/109th Annual Meeting of

the American-Gastroenterological-Association San Diego, CA, USA May 17

-22, 2008; 20080517

SPONSOR: Amer Gastroenterol Assoc

ISSN: 0016-5085

DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Background: Adiponectin is a peptide secreted from adipocytes that has insulin-sensitizing effects, is pro-apoptotic, and is

anti-inflammatory. Low circulating levels of adiponectin are associated with obesity, and have been associated with a number of epithelial cancers. A small pilot study suggested that low levels may be

associated with Barrett's esophagus (BE). The high molecular weight (HMW)

multimers of adiponectin are believed to be responsible for its insulin-sensitizing effect, but the role of the various multimers in carcinogenesis is largely unknown. We sought to examine the relationship

of adiponectin with BE, including the relationship of the HMW multimers. Methods: We performed a clinic-based, cross-sectional study of

risk factors for the presence of BE. Controls were subjects undergoing

elective upper endoscopy for classic gastroesophageal reflux symptoms

(GER), who did not harbor endoscopic or histological evidence of BE. Cases were subjects with endoscopic evidence of BE and histological

confirmation of intestinalized metaplasia. The levels of plasma adiponectin (total and HMW) was measured using a commercially available ELISA kit (ALPCO Diagnostics, Salem NH). Adiponectin levels (total, HMW, and non-HMW) were compared between cases and controls using logistic regression. Adjustments were made for age, gender, race, smoking, and the presence of a hiatal hernia. Results: Plasma samples were obtained from 116 cases of BE, and 234 GER controls. Among controls, total, HMW, and non-HMW adiponectin were inversely correlated with body mass index ($r = -0.24$, $p < 0.001$; $r = -0.20$, $p = 0.003$; $r = -0.23$, $p < 0.001$, respectively), and torso/buttocks ratio ($r = -0.22$, $p = 0.001$; $r = -0.17$, $p = 0.02$ $r = -0.22$, $p = 0.002$, respectively). Lower levels of total adiponectin were only marginally associated with BE [1st tertile vs. 3rd tertile: unadjusted odds ratio (uOR) 1.36, 95% confidence interval (CI) 0.79, 2.36; adjusted odds ratio (aOR) 1.21, 95% CI 0.60, 2.45]. Higher levels of HMW adiponectin were also marginally associated with BE (3rd tertile vs. 1st tertile: uOR 1.57, 95% CI 0.88, 2.79, aOR 1.67, 95% CI 0.82, 3.38). Lower levels of non-HMW adiponectin were strongly associated with BE compared to GER controls (1st tertile vs. 3rd tertile: uOR 4.79, 95% CI 2.48, 9.23, aOR 4.30, 95% CI 1.93, 9.60; 2nd tertile vs. 3rd tertile: uOR 2.47, 95% CI 1.23, 4.95, aOR 2.13, 95% CI 0.96, 4.74). Conclusions: Low circulating levels of non-HMW adiponectin is strongly associated with the presence of BE compared to GER controls. Adiponectin may be active in the pathogenesis of BE, and may be useful in risk stratification for BE among those with GER symptoms.

3/7/12 (Item 12 from file: 5)
 DIALOG(R)File 5:Biosis Previews(R)
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0020298117 BIOSIS NO.: 200800345056
 Metabolic profile in sons of women with polycystic ovary syndrome
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MAY 2008 2008
ITEM IDENTIFIER: doi:10.1210/jc.2007-2256
ISSN: 0021-972X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Context: Polycystic ovary syndrome (PCOS) is a common endocrine-metabolic disorder with strong familial aggregation. It has been demonstrated that parents and brothers of PCOS women exhibit insulin resistance and related metabolic defects. However, metabolic phenotypes in sons of PCOS women have not been described.

Objective: Our

objective was to assess the metabolic profiles in sons of women with PCOS

during different stages of life: early infancy, childhood, and adulthood.Design: Eighty sons of women with PCOS (PCOSS) and 56

sons of

control women without hyperandrogenism (C-S), matched for age, were studied. In early infancy, glucose and insulin were determined in the

basal sample. In children and adults, a 2-h oral glucose tolerance test

was performed with measurements of glucose and insulin. Adiponectin, leptin, C-reactive protein, SHBG, and serum lipids were determined in

the basal sample during the three periods.Results: During early infancy,

PCOSS showed higher weight ($P = 0.038$) and weight SD score ($P = 0.031$)

than C-S. During childhood, weight ($P = 0.003$), body mass index (BMI) (P

< 0.001), BMI SD score ($P < 0.001$), waist circumference ($P = 0.001$), total cholesterol ($P = 0.007$), and low-density lipoprotein cholesterol (P

$= 0.022$) were higher in PCOSS compared with C-S, but after adjusting for

BMI, these differences were nonsignificant. During adulthood, PCOSS exhibited higher weight ($P = 0.022$), BMI ($P = 0.046$), and waist circumference ($P = 0.028$) than C-S. Fasting insulin ($P = 0.030$), homeostasis model assessment for insulin resistance ($P = 0.034$), total

cholesterol ($P = 0.043$), low-density lipoprotein cholesterol ($P = 0.034$),

and 2-h insulin ($P = 0.006$) were also significantly higher and insulin

sensitivity index composite significantly lower in PCOSS than in C-S ($P =$

0.003). After adjusting for BMI, only 2-h insulin and insulin sensitivity index composite remained significantly different. Conclusions: This study indicates that sons of PCOS women exhibit higher body weight from early infancy. In addition, insulin resistance became evident as the subjects got older, which may place them at risk for the development of type 2 diabetes and cardiovascular disease.

3/7/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0020201323 BIOSIS NO.: 200800248262
Adiponectin multimer distribution, not absolute amount of plasma, correlates with depression severity in healthy elderly subjects
AUTHOR: Narita Kosuke; Murata Tetsuhito (Reprint); Hamada Toshihiko; Takahashi Tetsuya; Kosaka Hirotaka; Sudo Satoru; Mizukami Kimiko; Yoshida Haruyoshi; Wada Yuji
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LANGUAGE: English

ABSTRACT: Adiponectin is an adipocyte-specific secretory protein that circulates in serum as three oligomeric complexes known as the high, medium and low molecular weight form (HMW, MMW and LMW). HMW adiponectin has been suggested to be a better predictor of metabolic variables, and it was recently reported that the ratio of HMW to total adiponectin or to LMW, not the absolute amount of plasma adiponectin, might be crucial in determining insulin sensitivity. Insulin resistance (IR) is considered to be a primary component of vascular risk factors. Although the association of depression with atherosclerotic vascular diseases has been well documented, the contribution of IR to the evolution and progression of depression-associated vascular morbidity and mortality remains unknown.
The current preliminary study showed that the ratio of HMW to total

adiponectin or to LMW, not the absolute amount of plasma adiponectin, was negatively associated with depression severity in healthy elderly subjects without metabolic syndrome. This pilot study

supports a promising role of adiponectin multimer distribution for clarifying the pathophysiological mechanism by which

depression is associated with increased risk for IR, leading to cardiovascular disease, metabolic syndrome or type 2 diabetes. (c) 2007

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0020194083 BIOSIS NO.: 200800241022

Vascular effects of adiponectin: molecular mechanisms and potential therapeutic intervention

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DOCUMENT TYPE: Article; Literature Review

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LANGUAGE: English

ABSTRACT: Adiponectin is a major adipocyte-secreted adipokine abundantly present in the circulation as three distinct oligomeric complexes. In addition to its role as an insulin sensitizer, mounting

evidence suggests that adiponectin is an important player in maintaining vascular homoeostasis. Numerous epidemiological studies based

on different ethnic groups have identified adiponectin deficiency (hypo adiponectinaemia) as an independent risk factor for endothelial dysfunction, hypertension, coronary heart disease, myocardial infarction

and other cardiovascular complications. Conversely, elevation of circulating adiponectin concentrations by either genetic or pharmacological approaches can alleviate various vascular dysfunctions in

animal models. Adiponectin exerts its vasculoprotective effects through its direct actions in the vascular system, such as increasing

endothelial NO production, inhibiting endothelial cell activation and endothelium-leucocyte interaction, enhancing phagocytosis, and suppressing macrophage activation, macrophage-to-foam cell transformation and platelet aggregation. In addition, adiponectin reduces neointima formation through an oligomerization-dependent inhibition of smooth muscle proliferation. The present review highlights recent research advances in unveiling the molecular mechanisms that underpin the vascular actions of adiponectin and discusses the potential strategies of using adiponectin or its signalling pathways as therapeutic targets to combat obesity-related metabolic and vascular diseases.

3/7/15 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0020151494 BIOSIS NO.: 200800198433
High-molecular adiponectin as a marker of coronary artery disease
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DOCUMENT TYPE: Meeting; Meeting Abstract
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LANGUAGE: English

ABSTRACT: Background: Adiponectin is an adipocyte-specific secretory protein that is highly and specifically expressed in adipose tissue. Plasma adiponectin. level is decreased in obese individuals, to be negatively correlated with visceral fat accumulation and the lower level of adiponectin has been suggested to be associated with coronary artery disease, especially with the development of acute coronary syndrome. In human plasma, adiponectin circulates as a trimer, a hexamer and a high-molecular weight multimer. High-molecular weight (HMW) adiponectin of 420kDa is suggested to be more important as to vascular protective activities than total amount of adiponectin. However, clinical significance of the plasma HMW adiponectin in cooronary artery disease is not evident. Methods and Results: We measured HMW adiponectin level in 149 patients undergoing diagnostic coronary angiography, suspected of chronic coronary artery disease. The

high molecular adiponectin level was lower in patients with vasospastic angina ($3.4 \pm 2.4 \mu\text{g/dl}$, $P < 0.01$), stable effort angina (3.3 ± 2.6 , $P < 0.001$) and old myocardial infarction (3.8 ± 2.9 , $P < 0.01$), compared to chest pain syndrome patients (control) (6.6 ± 5.4). The level was lower in patients with multi-vessel disease ($3.4 \pm 2.4 \mu\text{g/dl}$), compared to patients with single vessel disease (4.2 ± 2.7 , $P < 0.05$) or no organic stenosis (5.1 ± 4.5 , $P < 0.01$). During observation of 7 years' follow-up, onset of cardiovascular events was seen in 50 patients (34%). Among various risk factors, diabetes ($P = 0.02$), insulin resistance assessed by homeostasis model assessment ($P = 0.06$), no statin use ($P = 0.08$), high sensitive C reactive protein level ($P = 0.0012$) and HMW adiponectin level ($P = 0.0037$) could predict cardiovascular events in univariate logistic regression analysis. However, multiple logistic regression analysis using these parameters showed that only HMW adiponectin was an independent predictor of cardiovascular event (OR; 2.23, 95%CI; 1.06-4.69, $P = 0.035$). Conclusion: HMW adiponectin may be not only a marker for severity of coronary artery disease but also a predictor of future cardiovascular events in patients with coronary artery disease.

3/7/16 (Item 16 from file: 5)
DIALOG(R)File 5:BIOSIS Previews(R)
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0020148187 BIOSIS NO.: 200800195126
A comparative study of the prevalence of the metabolic syndrome and its components in type 2 diabetic patients in two Caribbean islands using the new International Diabetes Federation definition
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OCT-DEC 2007 2007
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ISSN: 1381-3455

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background and aim: Tobago and Trinidad are two Caribbean islands with distinct genetic background and lifestyles; while Tobago is serene and a tourist centre, Trinidad is characterized by a hustling and bustling lifestyle. The study was aimed at determining and comparing the prevalence of the metabolic syndrome (MetS) and its critical components in type 2 diabetic patients using the new International Diabetes Federation (IDF) definition. Methods: Four hundred and thirteen (166 Tobago, 247 Trinidad) type 2 diabetic patients visiting 10 lifestyle disease clinics were studied. Blood pressure, anthropometric parameters (height, weight, body mass index and waist circumference) and overnight fasting blood samples were taken. Plasma glucose and serum triglycerides, total cholesterol, LDL- and HDL-cholesterol, insulin, and adiponectin were determined. Insulin resistance (IR) was determined using-the HOMA method. Results: The patients in Tobago were significantly older than patients in Trinidad ($p < 0.001$) but the duration of diabetes (9.4 ± 0.5 vs. 11.1 ± 0.7 yr), medications, generalized (31.7 vs. 38.8%) and central (78.5 vs. 83.7%) obesity were similar ($p > 0.05$). In comparison with patients in Tobago, diabetic patients in Trinidad, irrespective of gender, had significantly higher prevalence of IDF critical components such as raised BP, raised triglycerides and reduced HDL-cholesterol (all, $p < 0.001$). Thus, while more patients in Trinidad were diagnosed with MetS based on three or four components, more patients in Tobago were diagnosed based on two components ($p < 0.001$). Conclusions: There were high prevalence rates of the components of the MetS in both the islands of Tobago and Trinidad. Quantitatively, the aggregation of the components is higher in patients in Trinidad, which constitute greater risk for adverse cardiovascular outcome. Controlling central obesity should be the target in preventing MetS in the two islands.

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0020057647 BIOSIS NO.: 200800104586

High molecular weight multimer form of adiponectin as a useful
marker to evaluate insulin resistance and metabolic syndrome in
Japanese
men

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ABSTRACT: Adiponectin is an adipocyte-specific secretory protein that
possesses antidiabetic and anti atherosclerotic properties. Recent
studies have demonstrated that the high molecular weight (HMW)

multimer

form is the active form of this protein. In patients with type 2
diabetes mellitus, HMW-total adiponectin ratio was reported to be a
more useful marker than total adiponectin in the prediction of
insulin resistance and metabolic syndrome. In the present study of
healthy Japanese male subjects without any medication, we

investigated

the hypothesis that measuring only HMW adiponectin may be as
effective as HMW-total ratio to predict insulin resistance and/or
metabolic syndrome. This was a working community-based

cross-sectional

study of 637 male subjects aged 30 to 65 years. Total and HMW
adiponectin concentrations in serum were measured by enzyme-linked
immunosorbent assay using commercially available kits. Serum HMW
adiponectin level was inversely correlated with homeostasis model
assessment of insulin resistance (HOMA-IR) ($r = -0.375$, $P < .0001$)

even

after adjustment for age and body mass index ($r' = -0.245$, P
 $< .0001$).

When we divided the study subjects into quartile groups with equal
numbers of subjects, HOMA-IR in the 4 groups based on serum HMW
adiponectin level was significantly different ($P < .01$). Metabolic
syndrome score in the 4 groups based on serum HMW adiponectin level
was also significantly different ($P < .01$). Area under the curve of
receiver operator characteristic curves of HMW adiponectin (0.73)
to evaluate the presence of insulin resistance (HOMA-IR > 2.5) was
larger

than that of total adiponectin (0.68) or HMW-total ratio (0.70). Area under the curve of receiver operator characteristic curves of HMW adiponectin (0.70) to evaluate the presence of metabolic syndrome (body mass index-based modified criteria) was also larger than that of total adiponectin (0.65), but equal to that of HMW-total ratio (0.70). These results suggest that simply measuring HMW adiponectin may be as effective as HMW-total ratio to evaluate the presence of insulin resistance and metabolic syndrome, at least in nondiabetic subjects who are not receiving any medication. (C) 2007 Elsevier Inc. All rights reserved.

3/7/18 (Item 18 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0020026143 BIOSIS NO.: 200800073082
Multimers and adiponectin gene 276G > T polymorphism in the Japanese population residing in rural areas
AUTHOR: Ishibashi Kiyomi; Takamura Noboru (Reprint); Aoyagi Kiyoshi; Yamasaki Hironori; Abiru Norio; Nakazato Mio; Kamihiro Shimeru; Maeda Takahiro
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ABSTRACT: Background: Although it has been shown that high-molecular weight adiponectin is an active form, few studies have attempted to clarify the relationship between high molecular weight adiponectin and markers linked with cardiovascular diseases in the general population. Methods: We screened 236 Japanese study participants recruited from the general population, residing in one large and four small islands. In addition to serum lipids and lipoproteins, serum total adiponectin and each multimer were measured. The genotype single-nucleotide polymorphism 276G > T was detected in real-time PCR with LightCycler (R) hybridization probes, using fluorescent-labeled

nucleotides. Results: Multiple linear regression analysis showed that high-molecular weight adiponectin, as well as total adiponectin, were significantly correlated with body weight, body mass index, high-density lipoprotein cholesterol and triglycerides. Total adiponectin and high-molecular weight adiponectin concentrations were not significantly different between GG and TX (GT and TT) genotypes of 276G > T polymorphism in the adiponectin gene. Interestingly, no differences were observed for participants from the large island between GG and TX genotypes with regard to both total adiponectin and high-molecular weight adiponectin, whereas significant differences were observed for those from the small islands. Conclusions: Our results show that total adiponectin and high-molecular weight adiponectin are associated with similar factors in the general population. Furthermore, different effects of 276G > T for participants from small and large islands suggest that regional background due to geographic barriers may control the effects of 276G > T on adiponectin concentrations.

3/7/19 (Item 19 from file: 5)
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0019999928 BIOSIS NO.: 200800046867
Total and high molecular weight but not trimeric or hexameric forms of adiponectin correlate with markers of the metabolic syndrome and liver injury in Thai subjects
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JOURNAL: Journal of Clinical Endocrinology & Metabolism 92 (11): p 4313-4318 NOV 2007 2007
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LANGUAGE: English

ABSTRACT: Context/Objective: Decreased total adiponectin has been associated with metabolic disorders, including obesity, diabetes, fatty liver, and the metabolic syndrome. Although circulating adiponectin is composed of trimers, hexamers, and high molecular weight (HMW)

multimers, there has been limited study of the specific metabolic correlates of these isoforms in humans. Thus, our objective was to evaluate the associations of these adiponectin isoforms with metabolic and anthropometric parameters.

Design/Participants/Setting: A total of 53 diabetic and 68 nondiabetic subjects attending outpatient clinics underwent cross-sectional metabolic characterization.

Circulating levels of HMW, hexameric, and trimeric adiponectin were measured using a multimeric adiponectin ELISA based upon selective protease-mediated digestion.

Results: On Spearman univariate analysis, both total and HMW adiponectin levels were inversely associated with body mass index, fasting glucose, homeostasis model of assessment of insulin resistance, triglycerides, and alanine aminotransferase (ALT) (all vertical bar r vertical bar ≥ 0.22 ; $P < 0.05$), with the HMW isoform also positively correlated with high-density lipoprotein cholesterol ($r = 0.19$; $P = 0.036$). In contrast, hexameric and trimeric adiponectin were significantly associated with only body mass index ($r = -0.23$; $P = 0.0102$) and mid-upper arm circumference ($r = 0.21$; $P = 0.039$), respectively. On separate forward stepwise multiple linear regression analyses, fasting glucose and ALT emerged as independent, negative covariates of both total and HMW adiponectin, whereas no independent covariates of hexameric and trimeric adiponectin were identified. Furthermore, after adjustment for age, gender, and diabetes, mean ALT was highest in subjects in the lowest tertile of HMW adiponectin, followed in turn by the middle and highest tertiles, respectively (trend $P = 0.028$).

Conclusions: HMW adiponectin, but not hexameric or trimeric, tracks with the metabolic correlates of total adiponectin. Furthermore, an independent inverse association exists between ALT and HMW adiponectin.

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0019875706 BIOSIS NO.: 200700535447
Improved ELISA for selective measurement of adiponectin multimers and identification of adiponectin in human cerebrospinal fluid
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ABSTRACT: Background: Human serum adiponectin exists in 3
multimer forms: high molecular weight (HMW), middle molecular
weight, and low molecular weight (LMW), with some of the latter
bound to
albumin (Alb)-LMW. Some studies have suggested that adiponectin
crosses the blood-brain barrier and plays a central role in energy
homeostasis. Methods: To determine cerebrospinal fluid (CSF)
adiponectin at extremely low concentrations, we modified the
protocol of the ELISA system used to assay serum adiponectin. The 3
multimers of adiponectin were measured separately by pretreating
CSF with 2 proteases. We measured the CSF adiponectin
concentrations in anonymous human samples (n = 19). The molecular
sizes
of adiponectin in CSF pretreated with proteases or untreated were
determined by use of native PAGE and immunoblotting. Results: The
ELISA
system measured adiponectin in the range of 1.0-167 μ g/L. The
between-assay imprecision estimates (CVs) were 6%-17% for the 3
forms.
The mean total CSF adiponectin concentration (7.2 μ g/L) was
similar to 1/ 1000 of the mean concentration in serum. Unlike serum
adiponectin, the LMW and Alb-LMW forms predominated in all of the
CSF samples. Immunoblotting analysis revealed that most LMW forms
were
bound to Alb, although the HMW form was detected in some
samples. Conclusions: The modified ELISA system measures the 3
multimers
separately and is sufficiently sensitive to measure adiponectin in
CSF. (c) 2007 American Association for Clinical Chemistry.

3/7/21 (Item 21 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0019871880 BIOSIS NO.: 200700531621
Enhanced adiponectin multimer ratio and skeletal muscle
adiponectin receptor expression following exercise training and
diet in older insulin-resistant adults
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JOURNAL: American Journal of Physiology - Endocrinology and
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ITEM IDENTIFIER: doi:10.1152/ajpendo.00123.2007
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ABSTRACT: Circulating adiponectin is reduced in disorders associated with insulin resistance. This study was conducted to determine whether an exercise/diet intervention would alter adiponectin multimer distribution and adiponectin receptor expression in skeletal muscle. Impaired glucose-tolerant older (> 60 yr) obese (BMI 30-40 kg/m²) men ($n = 7$) and women ($n = 14$) were randomly assigned to 12 wk of supervised aerobic exercise combined with either a hypocaloric (ExHypo, similar to 500 kcal reduction, $n = 11$) or eucaloric diet (ExEu, $n = 10$). Insulin sensitivity was determined by the euglycemic, (5.0 mM) hyperinsulinemic (40 mU(.)m⁻² (.)min⁻¹) clamp. Adiponectin multimers [high (HMW), middle (MMW), and low molecular weight (LMW)] were measured by nondenaturing Western blot analysis. Relative quantification of adiponectin receptor expression through RT-PCR was determined from skeletal muscle biopsy samples. Greater weight loss occurred in ExHypo compared with ExEu subjects (8.0 \pm 0.6 vs. 3.2 \pm 0.6%, $P < 0.0001$). Insulin sensitivity improved postintervention in both groups (ExHypo: 2.5 \pm 0.3 vs. 4.4 \pm 0.5 mg(.)kg FFM⁻¹)min⁻¹, and ExEu: 2.9 \pm 0.4 vs. 4.1 \pm 0.4 mg(.)kg FFM⁻¹)min⁻¹, $P < 0.0001$). Comparison of multimer isoforms revealed a decreased percentage in MMW relative to HMW and LMW ($P < 0.03$). The adiponectin SA ratio (HMW/total) was increased following both interventions ($P < 0.05$) and correlated with the percent change in insulin sensitivity ($P < 0.03$). Postintervention adiponectin receptor mRNA expression was also significantly increased (AdipoR1 $P < 0.03$, AdipoR2 $P < 0.02$). These data suggest that part of the improvement in insulin sensitivity following

exercise and diet may be due to changes in the adiponectin oligomeric, distribution and enhanced membrane receptor expression.

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DIALOG(R)File 5:Biosis Previews(R)
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0019817901 BIOSIS NO.: 200700477642
Ethnic differences in adiponectin multimer distribution and relationships with metabolic syndrome traits
AUTHOR: Lara-Castro Cristina; Doud Erin C; Munoz Julian; Hunter Gary R;
Gower Barbara A; Garvey W Timothy
JOURNAL: Diabetes 56 (Suppl. 1): pA361 JUN 2007 2007
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0019760460 BIOSIS NO.: 200700420201
Adiponectin and the metabolic syndrome: mechanisms mediating risk for metabolic and cardiovascular disease
AUTHOR: Lara-Castro Cristina (Reprint); Fu Yuchang; Chung B Hong; Garvey W Timothy
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JOURNAL: Current Opinion in Lipidology 18 (3): p263-270 JUN 2007 2007
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LANGUAGE: English

ABSTRACT: Purpose of review Adiponectin is secreted exclusively by adipocytes, aggregates in multimeric forms, and circulates at high concentrations in blood. This review summarizes recent studies highlighting cellular effects of adiponectin and its role in human lipid metabolism and atherosclerosis. Recent findings Adiponectin is an important autocrine/paracrine factor in adipose tissue that modulates

differentiation of preadipocytes and favors formation of mature adipocytes. It also functions as an endocrine factor, influencing whole-body metabolism via effects on target organs. Adiponectin multimers exert differential biologic effects, with the high-molecular-weight multimer associated with favorable metabolic effects (i.e. greater insulin sensitivity, reduced visceral adipose mass, reduced plasma triglycerides, and increased HDL-cholesterol). Adiponectin influences plasma lipoprotein levels by altering the levels and activity of key enzymes (lipoprotein lipase and hepatic lipase) responsible for the catabolism of triglyceride-rich lipoproteins and HDL. It thus influences atherosclerosis by affecting the balance of atherogenic and antiatherogenic lipoproteins in plasma, and by modulating cellular processes involved in foam cell formation. Summary Recent studies emphasize the role played by adiponectin in the homeostasis of adipose tissue and in the pathogenesis of the metabolic syndrome, type 2 diabetes, and atherosclerosis. These pleiotropic effects make it an attractive therapeutic target for obesity-related conditions.

3/7/24 (Item 24 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0019679949 BIOSIS NO.: 200700339690
Selective purification and characterization of adiponectin multimer species from human plasma
AUTHOR: Hada Yusuke; Yamauchi Toshimasa; Waki Hironori; Tsuchida Atsushi;
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ITEM IDENTIFIER: doi:10.1016/j.bbrc.2007.03.004
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DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin is an adipocyte-derived hormone and known to form several species of multimer, however, the precise components

of each multimer have not been fully determined. We purified each multimer adiponectin selectively from human plasma and characterized them by affinity columns using anti-adiponectin, gelatin, or anti-albumin antibody and gel filtration. We found that adiponectin exists as four species of multimers in human plasma. According to their migrating mobility and N-terminal amino acid analysis,

we defined them as a trimer, albumin-binding trimer, hexamer, and HMW.

Low pH shifted HMW to hexamer, raising the possibility that HNIW is a 12

mer or larger multimer. We also showed that HMW had the highest binding activity to the membrane fractions of C2C12 myocytes and activated AMPK most potently. Our results indicate that adiponectin forms diverse multimer species and at least some of the functional properties are dependent on a multimer status. (c) 2007 Elsevier Inc. All rights reserved.

3/7/25 (Item 25 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0019598281 BIOSIS NO.: 200700258022
Adiponectin produced by lymphocytes inhibits granulopoiesis.
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JOURNAL: Blood 108 (11, Part 1): p374A NOV 16 2006 2006
CONFERENCE/MEETING: 48th Annual Meeting of the
American-Society-of-Hematology Orlando, FL, USA December 09 -12,
2006;
20061209
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ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Previous studies by our group have shown that normal unstimulated

lymphocytes produce a protein which inhibits colony formation of granulopoietic progenitors, but has no effect on erythroid progenitors.

Therefore, this inhibitor was initially designated GIA (granulopoietic

inhibitory activity). GIA was identified as a glycoprotein of approximately 30 kDa, with a pI of 7.9 - 8.4. Furthermore, we demonstrated

that this inhibitor may have physiological significance in that its production is altered in patients with neutropenia. GIA has proved

difficult to characterise to date since it is produced in relatively low amounts although it has a high specific biological activity. Adiponectin is an adipokine reported to share many of the inhibitory characteristics of GIA and has been demonstrated to act as a negative regulator of hematopoiesis and immune response. This study aimed to determine whether GIA is adiponectin or if it represents an adiponectin-like molecule. Lymphocyte conditioned medium (LCM) from lymphocytes cultured at 1×10^6 cells/ml in HL- I minimal medium was used as a source of GIA. Inclusion of LCM as 10% of the top layer of agar in a myeloid colony assay inhibited growth of CFU-GM by 52.11 % (n=3), confirming the presence of the inhibitory activity. RNA and protein from lymphocytes and LCM harvested over a 7 day culture period were subsequently investigated for adiponectin expression. Western blot analysis demonstrated a distinct banding pattern in days 3-7 LCM corresponding to monomers, dimers, trimers and greater. This is consistent with adiponectin which circulates as a multimer of trimers. Characterisation of GIA at the transcript level confirmed that GIA is in fact adiponectin. The N-terminal collagenous domain, C terminal globular domain and full length adiponectin were amplified by RT-PCR analysis. Adiponectin is thought to be secreted exclusively from adipocytes and much of our current knowledge of this molecule relates to its metabolic functions. Our study provides evidence that adiponectin is also produced by lymphocytes and may play a role in the pathogenesis of neutropenia.

3/7/26 (Item 26 from file: 5)
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0019587970 BIOSIS NO.: 200700247711
Adiponectin, ghrelin, and leptin differentially influence human platelet and human vascular endothelial cell functions: Implication in obesity-associated cardiovascular diseases
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JOURNAL: European Journal of Pharmacology 558 (1-3): p7-13 MAR 6
2007 2007
ISSN: 0014-2999
DOCUMENT TYPE: Article
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LANGUAGE: English

ABSTRACT: A very strong epidemiological link exists between obesity, the metabolic syndrome, diabetes and diabetes-associated cardiovascular pathologies. For this reason the peripheral effects of the centrally-acting satiety adipokines, adiponectin and leptin, and of non-adipose-derived hormones with similar effects, like ghrelin, have received considerable attention. In this report, we have extended our previous studies of the prothrombotic effects of leptin and determined the effects of adiponectin or ghrelin on human platelet activation. Thus, while leptin stimulated human platelet aggregation and adhesion, addition of adiponectin or of ghrelin did not affect either aggregation or adhesion of these cells; even at supraphysiological concentrations. In addition, we compared the impact of these three important hormones on microvascular endothelial cell permeability, an important parameter of endothelial function that when impaired contributes to several vascular pathologies. While physiologically relevant concentrations of either leptin or adiponectin increased the integrity of the diffusion barrier formed by a monolayer of human microvascular endothelial cells, only supra-physiological concentrations of ghrelin had this effect. None of these agents reduced microvascular endothelial barrier function. Taken together, our data are consistent with the ideas that leptin activates human platelets and limits transendothelial cell diffusion but that adiponectin only influences endothelial cell permeability. In contrast, ghrelin had neither of these effects. We propose that these data identify important differences in the effects of leptin, adiponectin or ghrelin on microvascular endothelial cells and platelets and may provide a basis on which to pharmacologically manipulate the selective effects of these peptides on these cell types in human cardiovascular or thrombotic diseases associated with obesity. (c)
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0019560653 BIOSIS NO.: 200700220394

Increased basal platelet activity, plasma adiponectin levels, and diabetes mellitus are associated with poor platelet responsiveness to in

vitro effect of aspirin

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JOURNAL: Thrombosis Research 119 (4): p517-524 2007 2007

ISSN: 0049-3848

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Introduction: Aspirin is one of the most effective antiplatelet

agents and is now commonly used to prevent vascular events. In some patients, however, recurrent vascular events have been demonstrated despite aspirin therapy. Our objective was to characterize individuals

showing poor response to in vitro effect of aspirin, using PFA-100. Methods: One hundred sixty-eight healthy male subjects were analyzed. We assessed platelet function tests, including PFA-100, whole

blood aggregation, and optical platelet aggregation. Also measured were hemostatic and other parameters including von Willebrand

factor (VWF:Ag), VWF ristocetin cofactor activity (VWF:RCo), soluble vascular adhesion molecule-1 (sVCAM-1), high sensitive C-reactive protein

(hs-CRP), and adiponectin. Poor responders were defined as having a collagen/epinephrine-induced closure time (CEPI-CT) under 250 s with PFA-100 when incubated with 10 μ M aspirin, whereas good responders were

defined as having a CEPI-CT of more than 250 s. Results and conclusions:

PFA-100 tests revealed that 40 subjects (24%) were poor responders (PR)

and 128 (76%) were good responders (GR). Poor responsiveness was significantly associated with (1) higher basal platelet activities in

PFA-100, as well as in whole blood aggregation and aggregometer; (2) increased level of adiponectin ($8.8 \pm 4.1 \mu$ g/mL [PR] vs $7.3 \pm$

2.9 μ g/mL [GR], $p=0.010$); and (3) the presence of diabetes mellitus (17.5% [PR] vs 4.7% [GR], $p=0.009$). Importantly, whereas 24% of the subjects showed insufficient inhibition in PFA-100 when incubated with 10

PM aspirin, almost all subjects showed maximum inhibition with 30 μ M

aspirin. These observations suggest that higher doses of aspirin might

overcome aspirin resistance. (c) 2006 Elsevier Ltd. All rights reserved.

3/7/28 (Item 28 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0019452627 BIOSIS NO.: 200700112368
A patient with Werner syndrome and adiponectin gene mutation
AUTHOR: Hashimoto Naotake (Reprint); Hatanaka Sachiko; Yokote Koutaro;
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JOURNAL: Diabetes Research and Clinical Practice 75 (1): p27-29 JAN
2007
2007
ISSN: 0168-8227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Werner syndrome is a premature aging disease characterized by
genomic instability and increased cancer risk. Here, we report a
45-year-old diabetic man as the first Werner syndrome patient found
to
have an adiponectin gene mutation. Showing graying and loss of
hair, skin atrophy, and juvenile cataract, he was diagnosed with
Werner
syndrome type 4 by molecular analysis. His serum adiponectin
concentration was low. In the globular domain of the adiponectin
gene, I164T in exon 3 was detected. When we examined effects of
pioglitazone (15 mg/day) on serum adiponectin multimer and
monomer concentrations using selective assays, the patient's
relative
percentage increased in adiponectin concentration was almost same
as that in the 18 diabetic patients without an adiponectin
mutation, but the absolute adiponectin concentration was half of
those seen in diabetic patients treated with the same pioglitazone
dose

who had no adiponectin mutation. The response suggested that pioglitazone treatment might help to prevent future Werner syndrome-related acceleration of atherosclerosis. Present and further

clinical relevant to atherosclerosis in this patient should be informative concerning the pathogenesis and treatment of atherosclerosis

in the presence of hypoadiponectinemia and insulin resistance. (c) 2006

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3/7/29 (Item 29 from file: 5)
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0019448154 BIOSIS NO.: 200700107895

Serum concentrations of adiponectin and characterization of adiponectin protein complexes in dogs

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JOURNAL: American Journal of Veterinary Research 68 (1): p57-62 JAN 2007

2007

ISSN: 0002-9645

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Objective-To assess serum concentrations of adiponectin and characterize adiponectin protein complexes in healthy dogs. Animals-11 healthy dogs. Procedures-Sera collected from 10 dogs were

evaluated via velocity sedimentation and ultracentrifugation, SDS-PAGE,

western immunoblotting, and radioimmunoassay, Visceral adipose tissue

(approx 90 g) was collected from the falciform ligament of a healthy dog

undergoing elective ovariohysterectomy, and adiponectin gene expression was assessed via a real-time PCR procedure. Results-

Adiponectin gene expression was detected in visceral adipose tissue. Serum adiponectin concentrations ranged from 0.85 to 1.5 mu g/ml (mean concentration, 1.22 mu g/mL). In canine serum,

adiponectin was present as a multimer, consisting of a low-molecular-weight complex (180 kd); as 3 (180-, 90-, and 60-kd) complexes under denaturing conditions; as 2 (90- and 60-kd)

complexes

under reducing conditions; and as a dimer, a monomer, and globular head region (60, 30, and 28 kd, respectively) under reducing-denaturing conditions. It is likely that adiponectin also circulates as a high-molecular-weight (360- to 540-kd) complex in canine serum, but resolution of this complex was not possible via SDS-PAGE. Conclusions and Clinical Relevance—After exposure to identical experimental conditions, adiponectin protein complexes in canine serum were similar to those detected in human and rodent sera. Circulating adiponectin concentrations in canine serum were slightly lower than concentrations in human serum. Adiponectin gene expression was identified in canine visceral adipose tissue. Results suggest that adiponectin could be used as an early clinical marker for metabolic derangements, including obesity, insulin resistance, and diabetes mellitus in dogs.

3/7/30 (Item 30 from file: 5)
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19410850 BIOSIS NO.: 200700070591
Circulating concentrations of high-molecular-weight adiponectin are increased following Roux-en-Y gastric bypass surgery
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JOURNAL: Diabetologia 49 (11): p2552-2558 NOV 2006 2006
ISSN: 0012-186X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Aims/hypothesis In addition to weight loss, bariatric surgery for severe obesity dramatically alleviates insulin resistance. In this study, we investigated whether circulating concentrations of the high-molecular-weight (HMW) form of adiponectin are increased following gastric bypass surgery. The HMW form is implicated as the multimer responsible for adiponectin's hepatic insulin-sensitising actions. Subjects and methods We studied 19 women who were undergoing Roux-en-Y gastric bypass surgery. Studies were conducted

prior to, and 1 and 12 months after surgery. Results One month after surgery, total plasma adiponectin concentrations were unchanged. Nevertheless, increases in both HMW (by 40 +/- 15%, p=0.006) and the proportion of adiponectin in the HMW form (from 40 +/- 2 to 50 +/- 2%, p < 0.0001) were observed. At 12 months, total and HMW adiponectin concentrations were increased by 58 +/- 8% and 118 +/- 21%, respectively (both p < 0.001). The majority (80%) of the increase of total adiponectin was due to an increase of the HMW form. After adjustment for covariates, increases of HMW and total adiponectin at 12 months were correlated with the decrease of fat mass (HMW, p=0.0076; total, p=0.0302). In subjects with improved insulin sensitivity at 12 months after surgery (n=18), the increase of HMW, but not that of total adiponectin, predicted the relative decrease of insulin resistance (HMW: p=0.0044; total: p=0.0775, after adjustment for covariates). Conclusions/interpretation These data suggest that the reduction of fat mass following gastric bypass surgery is an important determinant of the increase of HMW adiponectin concentrations, which in turn is associated with and may contribute to the resulting improvement of insulin sensitivity.

3/7/31 (Item 31 from file: 5)
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19407881 BIOSIS NO.: 200700067622
Increased hypothalamic 5-HT_{2A} receptor gene expression and effects of pharmacologic 5-HT_{2A} receptor inactivation in obese A(y) mice
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JOURNAL: Biochemical and Biophysical Research Communications 351 (4): p
1078-1082 DEC 29 2006 2006
ISSN: 0006-291X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Serotonin (5-hydroxytryptamine; 5-HT) 2A receptors contribute to the effects of 5-HT on platelet aggregation and vascular smooth muscle cell proliferation, and are reportedly involved in decreases in

plasma levels of adiponectin, an adipokine, in diabetic subjects. Here, we report that systemic administration of sarpogrelate, a 5-HT_{2A} receptor antagonist, suppressed appetite and increased hypothalamic pro-opiomelanocortin and cocaine- and amphetamine-regulated transcript, corticotropin releasing hormone, 5-HT_{2C}, and 5-HT_{1B} receptor gene expression. A(y) mice, which have ectopic expression of the agouti protein, significantly increased hypothalamic 5-HT_{2A} receptor gene expression in association with obesity compared with wild-type mice matched for age. Systemic administration of sarpogrelate suppressed overfeeding, body weight gain, and hyperglycemia in obese A(y) mice, whereas it did not increase plasma adiponectin levels. These results suggest that obesity increases hypothalamic 5-HT_{2A} receptor gene expression, and pharmacologic inactivation of 5-HT_{2A} receptors inhibits overfeeding and obesity in A(y) mice, but did not increase plasma adiponectin levels. (c) 2006 Elsevier Inc. All rights reserved.

3/7/32 (Item 32 from file: 5)
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19321165 BIOSIS NO.: 200600666560
Platelet activation is associated with hypoadiponectinemia and carotid atherosclerosis
AUTHOR: Shoji Takuhito; Koyama Hidenori (Reprint); Fukumoto Shinya; Maeno Takaaki; Yokoyama Hisayo; Shinohara Kayo; Emoto Masanori; Shoji Tetsuo; Yamane Takahisa; Hino Masayuki; Shioi Atsushi; Nishizawa Yoshiki
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JOURNAL: Atherosclerosis 188 (1): p190-195 SEP 2006 2006
ISSN: 0021-9150
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin, an adipokine secreted specifically from adipose tissue, has plurifunctions including antidiabetic, antiatherosclerotic, and antiinflammatory functions. Recently, platelet activation and the subsequent local inflammation have been implicated in progression of atherosclerosis. The aim of the study is to examine the interrelation among plasma adiponectin levels, platelet activation

status and quantitatively determined carotid atherosclerosis.
Subjects (n = 277) including 136 type 2 diabetic, 138 hypertensive, and 203 hypercholesterolemic patients participated in the study. Platelet activation was determined as percentage of polymorphonuclear cells (PMNs) or monocytes aggregated with platelets analyzed by CD41-positivity determined by whole-blood flow cytometry. PMN-platelet aggregates were significantly and positively associated with carotid atherosclerosis (intimal-medial thickness, IMT) with the interaction stronger than that of monocyte-platelet aggregates. Stepwise regression analyses revealed that PMN-platelet aggregates were the third strongest determinant of carotid IMT, with age and HbA 1 c stronger independent determinants. Simple and stepwise regression analyses of the factors associated with PMN-platelet aggregates revealed that HbA 1 c ($r = 0.423$), serum adiponectin levels ($r = -0.289$) and age ($r = -0.184$) were the three independent determinants. Thus, our data unveil novel link between hypoadiponectinemia and platelet activation. (c) 2005 Elsevier Ireland Ltd. All rights reserved.

3/7/33 (Item 33 from file: 5)
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19228177 BIOSIS NO.: 200600573572
A novel ELISA system for selective measurement of human adiponectin multimers by using proteases
AUTHOR: Ebinuma Hiroyuki (Reprint); Miyazaki Osamu; Yago Hirokazu; Hara Kazuo; Yamauchi Toshimasa; Kadowaki Takashi
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JOURNAL: Clinica Chimica Acta 372 (1-2): p47-53 OCT 2006 2006
ISSN: 0009-8981
DOCUMENT TYPE: Article
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LANGUAGE: English

ABSTRACT: Background: Adiponectin, an antiatherogenic adipocyte-derived protein exists in human blood as multiple isoforms-trimeric low molecular weight (LMW), albumin-binding LMW (Alb-LMW), hexameric middle molecular weight (MMW), and high molecular weight (HMW) forms. We developed a novel ELISA system to detect total

human adiponectin and the selective level of each adiponectin multimer for investigating the distribution of these levels in human blood. Methods: Two monoclonal antibodies that were raised against human adiponectin were used to construct a sandwich ELISA to measure adiponectin levels. Adiponectin multimers were selectively measured after sample pretreatment with two proteases that specifically digested the trimeric forms or both the hexameric and trimeric forms. Results: The ELISA had a dynamic range of 0.075-4.8 ng/ml. Intraassay variations (CV) were 5.3% (total adiponectin), 4.1% (MMW+HMW), and 3.3% (HMW). Comparison of the results of ELISA and quantitative western blot analysis of multimeric adiponectin in serum samples revealed good correlation (LMW+Alb-LMW, $r = 0.873$; MMW, $r = 0.907$; HMW, $r = 0.950$). Each of the three forms of adiponectin multimer levels closely correlated with total adiponectin levels in healthy subjects. Conclusions: This ELISA system can be used to further investigate the physiological roles of human adiponectin multimers. (c) 2006 Elsevier B.V. All rights reserved.

3/7/34 (Item 34 from file: 5)
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19192087 BIOSIS NO.: 200600537482
Adiponectin added into the plasma of healthy probands does not affect platelet aggregability
AUTHOR: Steiskal David; Proskova Jitka; Solichova Pavlina
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JOURNAL: Biomedical Papers (Olomouc) 150 (1): p89-90 JUL 2006 2006
ISSN: 1213-8118
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Six healthy non-obese probands without medical therapy and history of disease were tested. In all of them platelet aggregability with addition of human recombinant adiponectin in different concentrations (100; 75; 50 and 25 ng/l) were measured. It is concluded that increased level of adiponectin has no significant antiaggregation effect on platelets from individuals without hypoadiponectinemia.

3/7/35 (Item 35 from file: 5)
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19130379 BIOSIS NO.: 200600475774

Adiponectin multimerization is dependent on conserved lysines in the collagenous domain: Evidence for regulation of multimerization by alterations in posttranslational modifications

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JOURNAL: Molecular Endocrinology 20 (7): p1673-1687 JUL 2006 2006

ISSN: 0888-8809

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Adiponectin is a secreted, multimeric protein with insulin-sensitizing, antiatherogenic, and antiinflammatory properties.

Serum adiponectin consists of trimer, hexamer, and larger high-molecular-weight (HMW) multimers, and these HMW multimers appear to

be the more bioactive forms. Multimer composition of adiponectin appears to be regulated; however, the molecular mechanisms involved are unknown. We hypothesize that regulation of adiponectin multimerization and secretion occurs via changes in posttranslational modifications (PTMs). Although a structural role for

intertrimer disulfide bonds in the formation of hexamers and HMW multimers is established, the role of other PTMs is unknown. PTMs identified in murine and bovine adiponectin include hydroxylation of multiple conserved proline and lysine residues and glycosylation of

hydroxylysines. By mass spectrometry, we confirmed the presence of these

PTMs in human adiponectin and identified three additional hydroxylations on Pro71, Pro76, and Pro95. We also investigated the role

of the five modified lysines in multimer formation and secretion of recombinant human adiponectin expressed in mammalian cell lines. Mutation of modified lysines in the collagenous domain prevented formation of HMW multimers, whereas a pharmacological inhibitor of prolyl- and lysyl-hydroxylases, 2,2'-dipyridyl, inhibited formation of

hexamers and HMW multimers. Bacterially expressed human adiponectin displayed a complete lack of differentially modified isoforms and failed

to form bona fide trimers and larger multimers. Finally, glucose-induced

increases in HMW multimer production from human adipose explants correlated with changes in the two-dimensional electrophoresis profile of

adiponectin isoforms. Collectively, these data suggest that adiponectin multimer composition is affected by changes in PTM in response to physiological factors.

3/7/36 (Item 36 from file: 5)
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19126323 BIOSIS NO.: 200600471718

Olanzapine treatment is associated with reduced high molecular weight adiponectin in serum - A potential mechanism for olanzapine-induced insulin resistance in patients with schizophrenia

AUTHOR: Richards Ayanthi A; Hickman Ingrid J; Wang Amy Y-H; Jones Amanda L;

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JOURNAL: Journal of Clinical Psychopharmacology 26 (3): p232-237 JUN 2006

2006

ISSN: 0271-0749

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LANGUAGE: English

ABSTRACT: Treatment of schizophrenia with olanzapine and other atypical

antipsychotic agents is associated with insulin resistance and diabetes

mellitus. The mechanism for this is not understood. Adiponectin is an insulin-sensitizing cytokine secreted by adipocytes. It is present in

serum in multimers of varying size. Trimers and hexamers are referred to

as low molecular weight (LMW) adiponectin. Larger multimers (12-, 18-, and 24-mers) have been designated high molecular weight (HMW) adiponectin and seem responsible for the insulin-sensitizing action of this adipokine. The aim of this study was to examine total adiponectin and LMW and HMW multimers in serum from patients with schizophrenia treated with either olanzapine (n = 9) or other typical

antipsychotics (n = 9) and compare results with 16 healthy sex-, body

mass index-, and age-matched controls. The effects of olanzapine on adiponectin protein expression and secretion in in

vitro-differentiated primary human adipocytes were also examined. Patients receiving olanzapine had significantly lower total serum adiponectin as compared with those on conventional treatment and controls (5.23 +/- 1.53 ng/mL vs. 8.20 +/- 3.77 ng/mL and 8.78 +/- 3.8 ng/mL; $P < 0.05$ and $P < 0.01$, respectively). The HMW adiponectin was also reduced in patients on olanzapine as compared with the disease and healthy control groups (1.67 +/- 0.96 ng/mL vs. 3.87 +/- 2.69 ng/mL and 4.07 +/- 3.2 ng/mL; $P < 0.05$ for both). The LMW adiponectin was not different between patient groups ($P = 0.15$) but lower in patients on olanzapine as compared with controls (3.56 +/- 10.85 ng/mL vs. 4.70 +/- 1.4 ng/mL; $P < 0.05$). In vitro, short duration (up to 7 days) olanzapine exposure had no effect on total adiponectin expression or multimer composition of secreted protein. In summary, this study demonstrates a correlation between olanzapine treatment and reduced serum adiponectin, particularly HMW multimers. This may not be a direct effect of olanzapine on adipocyte expression or secretion of adiponectin. These observations provide insights into possible mechanisms for the association between olanzapine treatment and insulin resistance.

3/7/37 (Item 37 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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19072283 BIOSIS NO.: 200600417678
Identification of amino-terminal region of adiponectin as a physiologically functional domain
AUTHOR: Ujiie Hidetoshi; Oritani Kenji (Reprint); Kato Hisashi; Yokota Takafumi; Takahashi Isao; Maeda Tetsuo; Masaie Hiroaki; Ichii Michiko; Kamada Yoshihiro; Tamura Shinji; Kihara Shinji; Funahashi Tohru; Tomiyama Yoshiaki; Kanakura Yuzuru
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JOURNAL: Journal of Cellular Biochemistry 98 (1): p194-207 MAY 1 2006 2006
ISSN: 0730-2312
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin is an abundant adipose-specific protein, which acts as an anti-diabetic, anti-atherogenic, and anti-inflammatory adipokine. Although recent advances in the field of adiponectin have been made by the identification of adiponectin receptors and by the understanding about relationship between its multimerization and functions, detailed molecular background remains unclear. Our established anti-human adiponectin antibodies, ANOC 9103 and ANOC 9104, blocked some adiponectin functions Such as the growth inhibition of 13-lymphocytes on stromal cells and the inhibition of acetylated LDL uptake in macrophages, Suggesting that they may recognize important functional regions of adiponectin. As a result of epitope mapping based on the ability to bind to the deleted adiponectin Mutants, we identified that these antibodies recognize amino-terminal region of adiponectin before the beginning of the collagen-like domain. Notably, a peptide fragment (DQETTTQGPVLLPLPKGACTGWMA) corresponding to amino acid residues 1741 of human adiponectin could bind to restricted types of cells and block adiponectin-induced cyclooxygenase-2 gene expression and prostaglandin E-2 production in MS-5 stromal cells. Moreover, the deletion of its amino-terminal region reduced the abilities to inhibit not only collagen-induced platelet aggregation but also diet-induced hepatic steatosis. These data indicate that amino-terminal region of adiponectin is a physiologically functional domain and that a novel receptor, which recognizes amino-terminal region of adiponectin, may exist on some types of cells. Further investigations will contribute to the understanding of molecular mechanisms about adiponectin functions as well as to the designing of novel strategies for the treatment of patients with insulin-resistance, vascular dysfunction, and chronic inflammation.

3/7/38 (Item 38 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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19061038 BIOSIS NO.: 200600406433
A novel enzyme-linked immunosorbent assay specific for high-molecular-weight adiponectin
AUTHOR: Nakano Yasuko (Reprint); Tajima Sachiko; Yoshimi Ai; Akiyama Haruyo
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JOURNAL: Journal of Lipid Research 47 (7): p1572-1582 JUL 2006 2006
ISSN: 0022-2275

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Human plasma contains at least three forms of adiponectin: a trimer, a hexamer, and a high-molecular-weight (HMW) multimer. We purified HMW adiponectin from human plasma using its affinity to gelatin and obtained monoclonal antibodies against it. On Western blot analysis, the reactivity of these monoclonal antibodies was shown to be restricted to a non-heat-denatured form of adiponectin molecules. On heating, the collagen-like domain of adiponectin molecules became denatured, and thus the trimer form could not be maintained. From these, monoclonal antibodies against HMW adiponectin were suggested to react with the intact trimer of adiponectin. With these monoclonal antibodies, we developed a sandwich ELISA system for quantifying adiponectin in human serum. Its specificity was verified by analysis of serum fractions separated by gel-filtration chromatography, and our ELISA system was found to be HMW adiponectin-specific. With this novel ELISA, the HMW adiponectin concentrations were $8.4 \pm 5.5 \mu\text{g/ml}$ (mean \pm SD) in healthy women and $6.2 \pm 3.6 \text{ mg/ml}$ in healthy men. Also, serum with a lower HMW adiponectin concentration was shown to have a lower HMW ratio (i.e., HMW adiponectin/total adiponectin).

3/7/39 (Item 39 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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18816017 BIOSIS NO.: 200600161412
Adiponectin multimeric complexes and the metabolic syndrome trait cluster
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JOURNAL: Diabetes 55 (1): p249-259 JAN 2006 2006
ISSN: 0012-1797
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin circulates in human plasma mainly as a 180-kDa low molecular weight (LMW) hexamer and a high molecular weight (HMW) multimer of similar to 360 kDa. We comprehensively examined the

relationships between circulating levels of total adiponectin, adiponectin multimers, and the relative distribution (i.e., ratio) of multimeric forms with key features of the metabolic syndrome.

Total

adiponectin ($r = 0.45$), HMW ($r = 0.47$), LMW ($r = 0.31$), and HMW-to-total adiponectin ratio ($r = 0.29$) were significantly correlated with insulin-stimulated glucose disposal rate. Similarly, total ($r = -0.30$), HMW ($r = -0.38$), and HMW-to-total adiponectin ratio ($r = -0.34$) were correlated with central fat distribution but

not

with total fat mass or BMI. Regarding energy metabolism, although there

were no effects on resting metabolic rate, total ($r = 0.41$) and HMW ($r =$

0.44) were associated with increasing rates of fat oxidation.

HMW-to-total adiponectin ratio increased as a function of total adiponectin, and it was HMW quantity (not total or HMW-to-total adiponectin ratio or LMW) that was primarily responsible for all of these relationships. Impact on nuclear magnetic resonance

lipoprotein

subclasses was assessed. HMW and total adiponectin were correlated with decreases in large VLDL ($r = -0.44$ and -0.41); decreases in small

LDL ($r = -0.41$ and -0.36) and increases in large LDL ($r = 0.36$ and 0.30)

particle concentrations accompanied by increased LDL particle size ($r =$

0.47 and 0.39); and increases in large HDL ($r = 0.45$ and 0.37) and HDL

particle size ($r = 0.53$ and 0.47). Most of these correlations persisted

after adjustment for metabolic covariables. In conclusion, first, serum

adiponectin is associated with increased insulin sensitivity, reduced abdominal fat, and high basal lipid oxidation; however, it is HAM

quantity, not total or HMW-to-total adiponectin ratio, that is primarily responsible for these relationships. Second, reduced quantities

of HMW independently recapitulate the lipoprotein subclass profile associated with insulin resistance after correcting for glucose disposal

rate and BMI. Finally, HMW adiponectin is an important factor in explaining the metabolic syndrome.

3/7/40 (Item 40 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

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18813474 BIOSIS NO.: 200600158869

Adiponectin acts as an endogenous antithrombotic factor

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JOURNAL: Arteriosclerosis Thrombosis and Vascular Biology 26 (1): p224-230

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LANGUAGE: English

ABSTRACT: Objective: Obesity is a common risk factor in insulin resistance

and cardiovascular diseases. Although hypoadiponectinemia is associated

with obesity-related metabolic and vascular diseases, the role of adiponectin in thrombosis remains elusive. Methods and Results: We investigated platelet thrombus formation in adiponectin knockout (APN-KO) male mice (8 to 12 weeks old) fed on a normal diet. There was no

significant difference in platelet counts or coagulation parameters between wild-type (WT) and APN-KO mice. However, APN-KO mice showed an

accelerated thrombus formation on carotid arterial injury with a He-Ne

laser (total thrombus volume: $13.36 \pm 4.25 \times 10^7$ arbitrary units for

APN-KO and $6.74 \pm 2.87 \times 10^7$ arbitrary units for WT; $n = 10$; $P < 0.01$). Adenovirus-mediated supplementation of adiponectin attenuated the enhanced thrombus formation. In vitro thrombus

formation on a type I collagen at a shear rate of 250 s^{-1} , as well as platelet

aggregation induced by low concentrations of agonists, was enhanced in APN-KO mice, and recombinant adiponectin inhibited the enhanced platelet aggregation. In WT mice, adenovirus-mediated overexpression of adiponectin additionally attenuated thrombus formation. Conclusion: Adiponectin deficiency leads to enhanced thrombus formation and platelet aggregation. The present study reveals a new role of adiponectin as an endogenous antithrombotic factor.

3/7/41 (Item 41 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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18672568 BIOSIS NO.: 200600017963
Adiponectin multimer ratio is increased following exercise and
diet treatment in impaired glucose tolerance
AUTHOR: Nardi Ashley E (Reprint); Marchetti Christine M; Phillips
Susan A;
Ciaraldi Theodore P; Kirwan John P
JOURNAL: Diabetes 54 (Suppl. 1): pA267 2005 2005
CONFERENCE/MEETING: 65th Annual Meeting of the
American-Diabetes-Association San Diego, CA, USA June 10 -14, 2005;
20050610
SPONSOR: Amer Diabet Assoc
ISSN: 0012-1797
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

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18574272 BIOSIS NO.: 200510268772
Enhanced platelet aggregation and thrombogenic tendency in
adiponectin-deficient mice
AUTHOR: Kato Hisashi (Reprint); Kashiwagi Hirokazu; Shiraga Masamichi;
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JOURNAL: Blood 104 (11, Part 1): p228A-229A NOV 16 2004 2004
CONFERENCE/MEETING: 46th Annual Meeting of the
American-Society-of-Hematology San Diego, CA, USA December 04 -07,
2004;
20041204
SPONSOR: Amer Soc Hematol
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin is a 30 kDa protein secreted specifically from
adipocytes and structurally composed of two distinct domains,
C-terminal
collagen-like domain andN-terminal complement C1q-like globular
domain.

Adiponectin is abundantly present in plasma at high concentration ranging from 2 to 30 μ g/ml. The plasma levels of adiponectin decreased in patients with obesity and diabetes. Recently it has been

demonstrated that adiponectin has an anti-atherogenic activity. Hypoadiponectinemia is an independent risk factor for coronary artery

disease in men. However, the role of adiponectin in hemostasis and thrombosis still remains obscure. In this study, we examined its role in

hemostasis and thrombosis using adiponectin-deficient (APN-KO) mice (Nat. Med. 2002 Maeda et al.). APN-KO mice were fed by normal chow and

studied at 8-12 weeks old. There were no differences in platelet counts,

PT, APTT and plasma fibrinogen levels between APN-KO and Wild-Type mice.

Neither Wild-Type nor APN-KO mice showed detectable atherosclerotic lesion in carotid artery as well as whole aorta. We examined tail-bleeding

times as a measure of primary hemostasis. The tail bleeding time was 96.9

+/- 34.9 seconds in APN-KO mice, which was shorter than that in wild type

mice (130.9 +/- 52.1 seconds, $n=30$, $p<0.05$). We next studied thrombus

formation in mice carotid artery using He-Ne laser induced in vivo thrombus formation model. Thrombus formation was induced by the interaction of irradiated He-Ne laser with Evans blue dye injected into

blood flow. The thrombus volumes formed during 10 minutes were significantly larger in APN-KO mice ($6.74 \pm 2.87 \times 10^7$ arbitrary units for wild-type v.s. $13.4 \pm 4.25 \times 10^7$ arbitrary units for APN-KO

mice, $n=10$, $p<0.01$). Adenovirus-mediated supplement of adiponectin compensated for the thrombotic tendency in APN-KO mice. In order to clarify the effects of adiponectin on platelet function, we performed ex vivo experiments. In platelet aggregation studies under stirring conditions using platelet-rich plasma, platelet aggregation induced by low concentrations of agonists (ADP 2.5 μ M, collagen 2.5 μ g/ml, PAR4 peptide 75 μ M) was enhanced in APN-KO mice. Again the adenovirus-mediated supplement of adiponectin compensated for the enhancement of platelet aggregation. We next studied the thrombus formation on collagen coated surface under flow conditions. The thrombus formation was enhanced in APN-KO mice under shear rate at 250 s⁻¹. Our data provide a first evidence that adiponectin plays a role in hemostasis and thrombosis as a negative modulator of platelet function.

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17990911 BIOSIS NO.: 200400361700

Effects of novel peptides derived from the acidic tail of synuclein (ATS)

on the aggregation and stability of fusion proteins

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JOURNAL: Protein Engineering Design & Selection 17 (3): p251-260
March

2004 2004

MEDIUM: print

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DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: The acidic tail of alpha-synuclein (ATSalpha) has been shown to

protect the glutathione S-transferase (GST)-ATSalpha fusion protein from

environmental stresses, such as heat, pH and metal ions. In this study,

we further demonstrated that the introduction of ATSalpha into other proteins, such as dehydrofolate reductase and adiponectin, renders the fusion proteins resistant to heat-induced aggregation and that the acidic tail of beta- or gamma-synuclein can also protect the fusion

proteins from heat-induced aggregation. Interestingly, the heat resistance of GST-ATSalpha deletion mutants, which contain shorter peptides derived from the highly charged regions of ATSalpha, was approximately proportional to the number of added Glu/Asp residues.

However, the negative charges in the ATSalpha-derived peptides appear

insufficient to explain the extreme heat resistance of the fusion proteins, since polyglutamates appeared to be much less effective than

the ATSalpha-derived peptides in conferring heat resistance on the fusion

proteins. These results suggest that not only the negatively charged residues but also the specific amino acid sequence of ATSalpha play an

important role in conferring extreme heat resistance on the fusion proteins. Furthermore, the heat-induced secondary structural changes and

thermal inactivation curves of GST-ATSalpha deletion mutants indicated

that the introduction of ATSalpha-derived peptides does not significantly affect the intrinsic stability of the fusion proteins.

3/7/44 (Item 44 from file: 5)
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17944881 BIOSIS NO.: 200400315638
Mechanisms of early insulin-sensitizing effects of thiazolidinediones in

type 2 diabetes

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JOURNAL: Diabetes 53 (6): p1621-1629 June 2004 2004

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LANGUAGE: English

ABSTRACT: Whereas thiazolidinediones (TZDs) are known to rapidly improve

insulin action in animals, short durations of TZD therapy have never been

studied in humans. Among the many known actions of TZDs, increased circulating levels of the high molecular weight (HMW) multimer of adiponectin may be an important insulin-sensitizing mechanism. We examined the effects of only 21 days of 45 mg of pioglitazone (P+) versus

placebo (P-) in nine subjects with type 2 diabetes (HbA1c, 10.9 +/- 0.6%;

BMI, 31.9 +/- 1.5 kg/m²). Total adiponectin levels increased by approximately twofold in P+ in association with increased adipose tissue

gene expression. However, plasma free fatty acid and glucose levels were

unchanged, and there were only minimal changes in other "adipokines."

Glucose fluxes ((3-3H)glucose infusion) were measured during 6-h euglycemic (5 mmol/l) "pancreatic clamp" studies

(somatostatin/glucagon/growth hormone) with stepped insulin levels. Pioglitazone induced marked decreases in endogenous glucose production

(P+ = 0.9 +/- 0.1 vs. P- = 1.7 +/- 0.3 mg . kg⁻¹ . min⁻¹; P < 0.05) at

physiologic hyperinsulinemia (50 μ U/ml), which was highly correlated with an increased ratio of HMW adiponectin/total levels ($r^2 = 0.90$). Maximal insulin stimulation (400 μ U/ml) revealed pioglitazone-associated increases in glucose T take ($P = 10.5 \pm 0.9$ vs. $P = 8.9 \pm 0.8$ mg \cdot kg $^{-1}$ \cdot min $^{-1}$; $P < 0.05$), which did not correlate with HMW or total adiponectin levels. Thus, only 21 days of pioglitazone therapy improved insulin action in humans with type 2 diabetes. Increased abundance of the HMW adiponectin multimer may contribute to the hepatic insulin-sensitizing effects of these agents.

3/7/45 (Item 45 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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17877926 BIOSIS NO.: 200400246873
Adiponectin concentrations as a criterion of metabolic control in persons with type 2 diabetes mellitus?
AUTHOR: Stejskal David (Reprint); Ruzicka Viktor (Reprint); Adamovska Sylva (Reprint); Jurakova Renata (Reprint); Proskova Jitka (Reprint); Jedelsky Ladislav (Reprint); Bartek Josef
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JOURNAL: Biomedical Papers (Olomouc) 147 (2): p167-172 December 2003
MEDIUM: print
ISSN: 1213-8118 (ISSN print)
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin (ADP) is an adipocytokine with many antiatherogenic properties; its decreased level is associated with numerous atherogenic diseases and syndromes (e.g. diabetes mellitus (DM), dyslipidemia, endothelial dysfunction, hypertension, and obesity). Decreased ADP values in blood may be an independent risk factor of atherosclerotic (ATS) complications. Aim of the study: 1) Do persons with type 2 diabetes have lower ADP values than individuals without DM but with a high risk of ATS complications? 2) Do ADP values differ between persons with well controlled and persons with uncontrolled type 2 diabetes? We examined 109 patients of the Metabolic Center of Hospital

Sternberk. Out of them, 58 had type 2 diabetes, others were individuals with variously expressed risk factors of early atherosclerosis (obesity, hypertension, age, family history, smoking, dyslipidemia, etc.). In all persons under this study the following parameters were determined in peripheral venous blood: adiponectin, resistin, leptin, ObRe, cholesterol, HDL-cholesterol, triacylglycerols, glucose, HbA1c, creatinine, urea, ALT, AST, CRP, homocysteine, thrombocyte aggregation after CPG induction. The whole group was divided according to the presence of type 2DM into two subgroups; persons with diabetes were divided into the well controlled and uncontrolled subgroups. All data obtained were processed statistically using the software SPSS for Windows and Medcalc. The adiponectin/BMI index correlated negatively with HbA1c value (correlation coefficient -0.37, $p = 0.00053$), triacylglycerols (-0.4, $p = 0.000001$), P-glucose (-0.3, $p = 0.0017$), uricemia (-0.35, $p = 0.0007$) and positively with HDL-cholesterol value (0.6, $p = 0.00001$). Women had higher adiponectin values than men. Persons with hypertension and with diabetes mellitus, individuals with atherogenic lipotype or persons with inflammation signs had lower values than individuals without these diseases and syndromes. Persons with wellcontrolled diabetes mellitus had higher values than persons with uncontrolled diabetes (medians of the adiponectin/BMI index 9.7 vs. 6.7, $p < 0.01$). Persons with type 2 diabetes mellitus have lower ADP values than persons with a high ATS risk without diabetes mellitus. Persons with wellcontrolled diabetes mellitus (DM) and with satisfactory compensation have significantly higher ADP levels (independently of other metabolic parameters of DM control). ADP may be a new marker of metabolic control in persons with a high risk of atherosclerotic complications.

3/7/46 (Item 46 from file: 5)
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17700865 BIOSIS NO.: 200400081622
Role of disulfide bonds in Acrp30/adiponectin structure and signaling specificity. Different oligomers activate different signal transduction

pathways.

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JOURNAL: Journal of Biological Chemistry 278 (50): p50810-50817
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LANGUAGE: English

ABSTRACT: Acrp30/adiponectin is an adipocyte-derived serum protein with important roles in regulation of lipid and glucose metabolism, but

which of its isoforms are biologically active remains controversial. We

addressed this issue by first characterizing the structure of each individual Acrp30 oligomer and the determinants responsible for multimer formation. Freeze etch electron microscopy showed the trimer to exhibit a ball-and-stick-like structure containing a large globular sphere, an extended collagen stalk, and a smaller sphere on the

opposite end of the stalk. The hexamer consists of two adjacent trimeric

globular domains and a single stalk composed of collagen domains from two

trimers. Although not necessary for trimer formation or stability, two of

the three monomers in an Acrp30 trimer are covalently linked by a disulfide bond between cysteine residues at position 22. In contrast,

assembly of hexameric and higher molecular weight (HMW) forms of Acrp30

depends upon formation of Cys22-mediated disulfide bonds because their

reduction with dithiothreitol or substitution of Cys22 with alanine led

exclusively to trimers. HMW and hexamer isoforms of Acrp30 activated NF-kappaB in C2C12 cells, but trimers, either natural, formed by reduction of Acrp30 hexamer, or formed by the C22A mutant, did not.

In

contrast, incubation of isolated rat extensor digitorum longus with naturally formed Acrp30 trimers or trimeric C22A Acrp30 led to increased

phosphorylation of AMP-activated protein kinase-alpha at Thr172 and its

activation. Hexameric and HMW Acrp30 could not activate AMP-activated

protein kinase. Thus, trimeric and HMW/hexameric Acrp30 activate different signal transduction pathways, and Acrp30 represents a novel

example of the control of ligand signaling via changes in its oligomerization state.

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DIALOG(R)File 5:Biosis Previews(R)
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17609575 BIOSIS NO.: 200300568294

Impaired multimerization of human adiponectin mutants associated with diabetes. Molecular structure and multimer formation of adiponectin.

AUTHOR: Waki Hironori; Yamauchi Toshimasa; Kamon Junji; Ito Yusuke; Uchida

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RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Adiponectin is an adipocyte-derived hormone, which has been shown to play important roles in the regulation of glucose and lipid metabolism. Eight mutations in human adiponectin have been reported, some of which were significantly related to diabetes and hypoadiponectinemia, but the molecular mechanisms of decreased

plasma

levels and impaired action of adiponectin mutants were not clarified. Adiponectin structurally belongs to the complement 1q family and is known to form a characteristic homomultimer. Herein, we

demonstrated that simple SDS-PAGE under non-reducing and non-heat-denaturing conditions clearly separates multimer species of adiponectin. Adiponectin in human or mouse serum and adiponectin expressed in NIH-3T3 or Escherichia coli formed a wide range of multimers from trimers to high molecular weight (HMW) multimers.

A disulfide bond through an amino-terminal cysteine was required for the

formation of multimers larger than a trimer. An amino-terminal Cys-Ser mutation, which could not form multimers larger than a trimer, abrogated the effect of adiponectin on the AMP-activated protein kinase pathway in hepatocytes. Among human adiponectin mutations, G84R and G90S mutants, which are associated with diabetes and hypoadiponectinemia, did not form HMW multimers. R112C and I164T mutants, which are associated with hypoadiponectinemia, did not assemble into trimers, resulting in impaired secretion from the cell. These data suggested impaired multimerization and/or the consequent impaired secretion to be among the causes of a diabetic phenotype or hypoadiponectinemia in subjects having these mutations. In conclusion, not only total concentrations, but also multimer distribution should always be considered in the interpretation of plasma adiponectin levels in health as well as various disease states.

3/7/48 (Item 48 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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17503204 BIOSIS NO.: 200300458815
Impaired multimerization of human adiponectin mutants associated with diabetes.
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JOURNAL: Diabetes 52 (Supplement 1): pA1 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 63rd Scientific Sessions of the American Diabetes Association New Orleans, LA, USA June 13-17, 2003; 20030613
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17379651 BIOSIS NO.: 200300336394

Clq-TNF Related Protein-1 (CTRP1) Prevents Thrombus Formation in Non-Human

Primates and Atherosclerotic Rabbits without Causing Bleeding.

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RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Injury to the arterial wall, often caused by rupturing atherosclerotic plaques, exposes pro-thrombotic agents including collagen

to circulating blood. As a consequence, circulating platelets adhere to

the site of injury and become fully activated, ultimately leading to thrombus formation that can trigger heart attack or stroke. The most effective anti-thrombotic therapies are designed to inhibit platelet function, possibly putting the patient at risk for severe bleeding complications. A less well-studied approach is to mask the binding sites

on collagen thereby inhibiting interaction with platelets. We discovered

a protein expressed in the vascular wall, CTRP1, which has a high binding

affinity for collagen. CTRP1 is a member of the Clq-TNF Related Protein

family that also includes Clq and Adiponectin. We hypothesized that CTRP1 could prevent platelet activation and thrombus formation by binding

to collagen. In vitro, CTRP1 inhibited platelet binding to collagen and

prevented platelet activation and aggregation dose-dependently. In vivo, CTRP1 was tested using a baboon arteriovenous (AV) shunt model and

a Folts vascular injury model. The AV-shunt model follows the accumulation of ¹¹¹In-labeled platelets on a collagen-coated graft. The

grafts were pretreated with CTRP1 or saline and then placed in the AV circuit for 60 minutes. Treatment with CTRP1 led to significantly reduced platelet accumulation ($1.86 \pm 1.00 \times 10^9$ vs. $0.28 \pm 0.32 \times 10^9$ platelets bound in 6 control and 5 treated animals respectively; $p=0.006$). Using the Folts model of cyclic flow variation in rabbits and cynomolgus monkeys, we demonstrated that treatment with CTRP1 abolished cyclic flow variations and maintained patency of injured, stenosed vessels. A further refinement of the Folts vascular injury model was achieved using rabbits with atherosclerotic lesions. Animals were placed on a high cholesterol diet for two weeks and then underwent balloon denudation of the right iliac artery. Three weeks after balloon injury, extensive atherosclerotic lesions covered the denuded region. A crush injury was made rupturing the atherosclerotic plaque and a stenosis was positioned over the injured segment. Once cyclic flow was established, animals were treated with 1.0 mg/kg CTRP1 or BSA. Treatment with CTRP1 resulted in reduction of platelet aggregation, increased blood flow, and maintained vessel patency. Unlike systemically acting platelet antagonists, CTRP1 had no effect on bleeding at therapeutic doses. This was demonstrated by following parameters of hemostasis as well as by performing template bleeds. In conclusion, CTRP1 is a potent anti-thrombotic protein that acts locally at the site of vascular injury by binding to collagen. Unlike conventional, systemically acting platelet antagonists it does not produce increased bleeding or a decrease in circulating platelet numbers.

3/7/50 (Item 50 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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17289432 BIOSIS NO.: 200300248151
Adiponectin is markedly increased in patients with nephrotic syndrome and is related to metabolic risk factors.
AUTHOR: Zoccali Carmine (Reprint); Mallamaci Francesca; Panuccio Vincenzo;
Tripepi Giovanni; Cutrupi Sebastiano; Parlongo Saverio; Catalano Francesco; Tanaka Sachiyo; Ouchi Noriyuki; Kihara Shinji; Funahashi Tohru

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JOURNAL: Kidney International 63 (Supplement 84): pS98-S102 May 2003
2003
MEDIUM: print
ISSN: 0085-2538 _(ISSN print)
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background. Adiponectin (ADPN), the gene product of apM1, is the most abundant secretory protein of the adipose tissue in human plasma. Altered regulation (reduced synthesis) of this substance may be relevant to endothelial dysfunction and cardiovascular complications in patients with ESRD. Methods. We investigated the relationship between plasma ADPN, glomerular filtration rate (GFR) (plasma iohexol clearance), and metabolic risk factors in 16 patients with nephrotic syndrome, in 25 patients with chronic nephropathies without nephrotic syndrome, and in 31 healthy subjects. Results. Plasma ADPN was much higher ($P < 0.01$) in patients with nephrotic syndrome (24.4 ± 14.9 mug/mL) than in patients with chronic nephropathies without nephrotic syndrome (12.3 ± 7.2 mug/mL) and healthy subjects (5.9 ± 2.6 mug/mL). In the aggregate 24-hour, proteinuria ($r = 0.53$, $P < 0.01$) and serum cholesterol ($r = 0.53$, $P < 0.01$) were strong and direct correlates of plasma ADPN, while serum albumin correlated inversely ($r = -0.46$, $P < 0.01$) with this protein. Proteinuria appeared to be an important confounder of the relationship between ADPN and the GFR because in the whole patient population (with and without nephrotic syndrome), this relationship emerged only after data adjustment for 24-hour proteinuria (partial $r = -0.31$, $P = 0.05$), while no such relationship was demonstrable on crude data analysis ($r = 0.03$, $P = 0.87$). Conclusions. ADPN is markedly increased in patients with nephrotic syndrome, and proteinuria is strongly related to circulating ADPN in patients with nephrotic and non-nephrotic renal diseases. The relationships between plasma ADPN, serum cholesterol, and serum albumin suggest that this adipocyte protein may serve to mitigate endothelial damage triggered by dyslipidemia and other risk factors in patients with chronic renal diseases.

3/7/51 (Item 1 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts
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0003622049 IP ACCESSION NO: 8907684
Globular adiponectin increases cGMP formation in blood platelets
independently of nitric oxide

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Journal of Thrombosis and Haemostasis, Suppl. 12, v 6, p 2121-2131,
December 2008
PUBLICATION DATE: 2008

PUBLISHER: Blackwell Publishing Ltd., 9600 Garsington Road

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 1538-7933
ELECTRONIC ISSN: 1538-7836
DOI: 10.1111/j.1538-7836.2008.03179.x
FILE SEGMENT: Calcium & Calcified Tissue Abstracts

ABSTRACT:

Summary.Background:Platelet-derived nitric oxide (NO) has been
shown to
play conflicting roles in platelet function, although it is accepted
that
NO mediates its actions through soluble guanylyl cyclase (sGC). This
confusion concerning the roles of platelet NO may have arisen because
of an
uncharacterized mechanism for activation of sGC. Objectives:To
examine the
ability of the novel platelet agonist globular adiponectin (gAd) to
stimulate the NO-independent cGMP-protein kinase G (PKG) signaling
cascade.
Methods:We used three independent markers of NO signaling,
[3H]l-citrulline
production, cGMP accrual, and immunoblotting of vasodilator-stimulated
phosphoprotein (VASP), to examine the NO signaling cascade in
response to
gAd. Results:gAd increased platelet cGMP formation, resulting in a
dose-
and time-dependent increase in phospho-VASP157/239. Phosphorylation
of VASP

in response to gAd was mediated by both protein kinase A and PKG. Importantly, cGMP formation occurred in the absence of NO synthase (NOS) activation and in the presence of NOS inhibitors. Indeed, inhibition of the NOS signaling cascade had no influence on gAd-mediated platelet aggregation. Exploration of the mechanism demonstrated that NO-independent cGMP formation, phosphorylation of VASP and association of sGC alpha 1 with heat shock protein-90 induced by gAd were blocked under conditions that inhibited Src kinases, implying a tyrosine kinase-dependent mechanism. Indeed, sGC alpha 1 was reversibly tyrosine phosphorylated in response to gAd, collagen, and collagen-related peptide, an effect that required Src kinases and downstream Ca²⁺ mobilization. Conclusions: These data demonstrate activation of the platelet cGMP signaling cascade by a novel tyrosine kinase-dependent mechanism in the absence of NO.

3/7/52 (Item 2 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts
(c) 2009 CSA. All rts. reserv.

0002989892 IP ACCESSION NO: 7205032
Increased hypothalamic 5-HT_{2A} receptor gene expression and effects of pharmacologic 5-HT_{2A} receptor inactivation in obese A super(y) mice

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1078-1082, December 29, 2006
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Netherlands, [mailto:nlinfo-f@elsevier.nl],
[URL:http://www.elsevier.nl/]

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LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 0006-291X
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FILE SEGMENT: CSA Neurosciences Abstracts

ABSTRACT:

Serotonin (5-hydroxytryptamine; 5-HT) 2A receptors contribute to the effects of 5-HT on platelet aggregation and vascular smooth muscle cell proliferation, and are reportedly involved in decreases in plasma levels of adiponectin, an adipokine, in diabetic subjects. Here, we report that systemic administration of sarpogrelate, a 5-HT_{2A} receptor antagonist, suppressed appetite and increased hypothalamic pro-opiomelanocortin and cocaine- and amphetamine-regulated transcript, corticotropin releasing hormone, 5-HT_{2C}, and 5-HT_{1B} receptor gene expression. A super(y) mice, which have ectopic expression of the agouti protein, significantly increased hypothalamic 5-HT_{2A} receptor gene expression in association with obesity compared with wild-type mice matched for age. Systemic administration of sarpogrelate suppressed overfeeding, body weight gain, and hyperglycemia in obese A super(y) mice, whereas it did not increase plasma adiponectin levels. These results suggest that obesity increases hypothalamic 5-HT_{2A} receptor gene expression, and pharmacologic inactivation of 5-HT_{2A} receptors inhibits overfeeding and obesity in A super(y) mice, but did not increase plasma adiponectin levels.

3/7/53 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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18880688 Genuine Article#: 408KA Number of References: 22
Title: Altered Molecular Weight Forms of Adiponectin in Hypertension
Author(s): Baumann M (REPRINT) ; von Eynatten M; Dan L; Richart T;
Kouznetsova T; Heemann U; Staessen JA
Corporate Source: Tech Univ Munich,Klinikum Rechts Isar, Dept
Nephrol,Ismaninger Str 22/D-81675 Munich//Germany/ (REPRINT);
Tech Univ
Munich,Klinikum Rechts Isar, Dept Nephrol,D-81675
Munich//Germany/;
Maastricht Univ,Dept Epidemiol, Genet Epidemiol
Unit,Maastricht//Netherlands/; Katholieke Univ Leuven,Dept
Cardiovasc
Dis, Div Hypertens & Cardiac Rehabil, Studies Coordinating
Ctr,Louvain//Belgium/
Journal: JOURNAL OF CLINICAL HYPERTENSION, 2009, V11, N1 (JAN), P11-16
ISSN: 1524-6175 Publication date: 20090100
Publisher: WILEY-BLACKWELL PUBLISHING, INC, COMMERCE PLACE, 350 MAIN
ST,

MALDEN 02148, MA USA

Language: English Document Type: ARTICLE

Abstract: An important link between adiponectin and hypertension has been proposed in clinical studies. In the circulation, adiponectin is predominantly present in multimeric complexes, of which high-molecular weight (HMW) adiponectin is thought to represent the biological active form. The authors investigated which

role the different multimeric adiponectin isoforms play in context with hypertension as compared to total adiponectin levels. Fifty (19 normotensive /31 hypertensive) patients were included in the study. Total adiponectin and adiponectin multimers were determined by enzyme-linked immunosorbent assay and western blot.

The authors analyzed associations between adiponectin multimer levels and blood pressure. Total adiponectin concentrations were not significantly different between hypertensive

and normotensive patients (6.8 ± 2.3 vs 7.5 ± 4.2 μ g/mL).

HMW

adiponectin was significantly lower ($P < .05$) and low-molecular weight adiponectin was significantly higher ($P < .01$) in hypertensive than in normotensive persons (3.8 ± 1.7 vs $5.2 \pm$

3.0

μ g/mL and 0.9 ± 0.5 vs 1.8 ± 0.9 , respectively). Low molecular

weight was an independent predictor for the presence of hypertension

(effect coefficient: 0.160-0.445; $P < .001$) in multivariate analyses.

These results suggest that the composition of the molecular weight forms of adiponectin in hypertension are characterized by reduced HMW adiponectin, the proposed major active form of adiponectin, and increased low-molecular weight adiponectin.

Moreover, the latter represents an independent predictor of prevalent

hypertension, suggesting an association between adiponectin multimer composition and hypertension. J Clin Hypertens

(Greenwich). 2009; 11: 11-16. (C) 2009 Wiley Periodicals, Inc.

3/7/54 (Item 2 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

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18804396 Genuine Article#: 400CF Number of References: 43

Title: Adipokines and Acute Coronary Syndrome

Author(s): Nakas-Icindic E (REPRINT) ; Valjevac A; Zaciragic A

Corporate Source: Univ Sarajevo, Fac Med, Inst Physiol & Biochem, Sarajevo

71000//Bosnia & Herceg/ (REPRINT); Univ Sarajevo, Fac Med, Inst Physiol

& Biochem, Sarajevo 71000//Bosnia & Herceg/

Journal: HEALTHMED, 2008, V2, N4, P225-233

ISSN: 1840-2291 Publication date: 20080000

Publisher: DRUNPP-SARAJEVO, BOLNICKA BB, SARAJEVO, 71000, BOSNIA & HERCEG

Language: English Document Type: ARTICLE

Abstract: Adipose tissue has traditionally been considered as a tissue devoted mainly to energy storage. Now it is recognized as a multifunctional organ involved in the production of hormones, growth factors and cytokines named adipokines.

In obese subject the production of adipokines is impaired, In obesity high level of leptin, resistin, and low level of adiponectin have been observed and implicated in insulin resistance, atherosclerosis and metabolic syndrome.

In patients with established coronary atherosclerosis increased

body weight is an independent predictor of all acute coronary syndrome.

The exact mechanism of obesity induced coronary heart disease is not

fully elucidated. Current research is aimed to determine links between

adipokines and coronary heart disease.

Leptin, adiponectin and resistin are adipokines that are implicated in coronary endothelial dysfunction, thrombogenesis and inflammation. These processes are known to precipitate atherosclerotic

plaque rupture and acute coronary syndrome.

Recent studies demonstrated that high plasma leptin and low adiponectin levels as observed in obese subjects impair coronary acetylcholine-mediated vasodilatation in vitro and in vivo.

Resistin

also impair coronary vasodilatation but via bradykinin pathway.

Leptin and resistin show proinflammatory effects upregulating cytokine production in macrophages and might lead to destabilization of

coronary atherosclerotic plaque. Leptin has been observed to stimulate

angiogenesis, platelet aggregation, and atherothrombosis in obese human. In obese subject the production of adiponectin, which has protective effects on coronary blood vessels is suppressed.

This paper summarizes the role of three adipokines: leptin, resistin and adiponectin in acute coronary syndrome and implicates their possible application in clinical practice.

3/7/55 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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18720900 Genuine Article#: 393ML Number of References: 43
Title: Leptin, but not adiponectin, is a predictor of recurrent
cardiovascular events in men: results from the LIPID study
Author(s): Soderberg S (REPRINT) ; Colquhoun D; Keech A; Yallop J;
Barnes
EH; Pollicino C; Simes J; Tonkin AM; Nestel P
Corporate Author(s): LIPID Study Investigators
Corporate Source: Umea Univ Hosp, Dept Publ Hlth & Clin Med, SE-90185
Umea//Sweden/ (REPRINT); Umea Univ Hosp, Dept Publ Hlth & Clin
Med, SE-90185 Umea//Sweden/; Univ
Queensland, Brisbane/Qld/Australia/;
Univ Sydney, Natl Hlth & Med Res Council Clin Trials Ctr, Sydney/NSW
2006/Australia/; Monash Univ, Dept Epidemiol & Prevent
Med, Melbourne/Vic
3004/Australia/; Natl Heart Fdn
Australia, Melbourne/Vic/Australia/;
Baker Heart Res Inst, Melbourne/Vic/Australia/
Journal: INTERNATIONAL JOURNAL OF OBESITY, 2009, V33, N1 (JAN),
P123-130
ISSN: 0307-0565 Publication date: 20090100
Publisher: NATURE PUBLISHING GROUP, MACMILLAN BUILDING, 4 CRINAN ST,
LONDON
N1 9XW, ENGLAND
Language: English Document Type: ARTICLE
Abstract: Objective: To investigate the relationships between plasma
leptin
and adiponectin levels and recurrent cardiovascular events
(cardiovascular death, nonfatal myocardial infarction and stroke)
in
men with earlier acute coronary syndromes.

Design, subjects and measurements: A nested case-control study
examined circulating leptin and adiponectin levels in plasma
obtained 4-6 years after entry into the Long-Term Intervention
with
Pravastatin in Ischaemic Disease (LIPID) trial. Plasma was
assayed from
184 men who suffered recurrent events within 4.4 years after blood
collection and 184 matched controls who remained free of further
events. The association between cardiovascular events and the
explanatory variables was examined by conditional logistic
regression
analysis.

Results: Relative risk (RR) increased across increasing leptin
quartiles; the highest quartile compared with the lowest quartile
was
related to the highest risk (P for trend = 0.002); the increased
risk

remained after adjustment for risk factors ($P = 0.018$) or for obesity ($P = 0.038$), but in the final model (adjusted for randomized treatment, other drugs, LIPID risk score, age and body mass index), the risk was attenuated ($RR = 1.61$, 95% CI: 0.72-3.57, P for trend = 0.34). Adiponectin did not predict cardiovascular events. Subjects randomly allocated to pravastatin had 6% lower leptin levels ($P = 0.04$) than those allocated to placebo.

Conclusion: Plasma leptin was a significant and independent predictor of recurrent cardiovascular events (cardiovascular death, nonfatal myocardial infarction and stroke) in men with earlier acute coronary syndromes.

3/7/56 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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18594513 Genuine Article#: 3790J Number of References: 44
Title: Adiponectin Multimers and Metabolic Syndrome Traits: Relative Adiponectin Resistance in African Americans
Author(s): Lara-Castro C (REPRINT) ; Doud EC; Tapia PC; Munoz AJ; Fernandez JR; Hunter GR; Gower BA; Garvey WT
Corporate Source: Univ Alabama, Dept Nutr Sci, Birmingham//AL/35294 (REPRINT)
; Univ Alabama, Dept Nutr Sci, Birmingham//AL/35294; Birmingham Vet Affairs Med Ctr, Birmingham//AL/
Journal: OBESITY, 2008, V16, N12 (DEC), P2616-2623
ISSN: 1930-7381 Publication date: 20081200
Publisher: NATURE PUBLISHING GROUP, 75 VARICK STREET, 9TH FLOOR, NEW YORK, NY 10013-1917 USA
Language: English Document Type: ARTICLE
Abstract: African Americans (AAs) tend to have lower total adiponectin levels compared to European Americans (EA); however, it is not known whether race affects adiponectin multimer distribution and their relationships to metabolic traits. We measured total adiponectin, high molecular weight (HMW), low molecular weight (LMW) (i.e., hexamer), and trimer adiponectin in 132 normoglycemic premenopausal women (75 AAs, 57 EAs), together with measures of total and abdominal fat, plasma lipids, insulin sensitivity (S-i), and genetic admixture estimates. We found that lower total adiponectin in AAs was explained by reduced LMW, and trimer forms because levels of HMW did not differ between races. In EAs, HMW was

highly correlated with multiple metabolic syndrome traits. In contrast, the LMW and trimer forms were most highly correlated with metabolic traits in AAs, including abdominal adiposity, lipids, and S-i. At similar levels of visceral adiposity, AAs exhibited significantly lower LMW adiponectin than EAs. Similarly, at comparable levels of HMW and LMW adiponectin, AAs were more insulin resistant than their EA counterparts. In conclusion, (i) serum adiponectin is lower in AAs predominantly as a result of reduced concentrations of LMW and trimers multimeric forms; (ii) LMW and trimer, not HMW, are most broadly correlated with metabolic traits in AAs. Thus, HMW adiponectin may exert less bioactivity in explaining the metabolic syndrome trait cluster in populations of predominant African genetic background.

3/7/57 (Item 5 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

18545398 Genuine Article#: 376GU Number of References: 268
Title: Adiponectin multimers in maternal plasma
Author(s): Mazaki-Tovi S; Romero R (REPRINT) ; Kusanovic JP; Erez O; Vaisbuch E; Gotsch F; Mittal P; Than GN; Nhan-Chang C; Chaiworapongsa T ; Edwin S; Camacho N; Nien JK; Hassan SS
Corporate Source: Hutzel Womens Hosp,Perinatol Res Branch, Intramural Div,
NICHD,NIH,DHHS,Box 4,3990 John R/Detroit//MI/48201 (REPRINT);
Hutzel
Womens Hosp,Perinatol Res Branch, Intramural Div,
NICHD,NIH,DHHS,Detroit//MI/48201; Hutzel Womens Hosp,Perinatol Res Branch, Intramural Div, NICHD,NIH,DHHS,Bethesda//MD/; Wayne State Univ,Hutzel Womens Hosp, Dept Obstet & Gynecol,Detroit//MI/; Wayne State Univ,Ctr Mol Med & Genet,Detroit//MI/; Univ Catolica Chile,Hosp
Sotero Rio, Ctr Perinatal Diag & Res CEDIP,Puente Alto//Chile/
Journal: JOURNAL OF MATERNAL-FETAL & NEONATAL MEDICINE, 2008, V21, N11, P 796-815
ISSN: 1476-7058 Publication date: 20080000
Publisher: TAYLOR & FRANCIS LTD, 4 PARK SQUARE, MILTON PARK, ABINGDON OX14 4RN, OXON, ENGLAND
Language: English Document Type: REVIEW
Abstract: Objective. Adiponectin is an anti-diabetic, anti-atherogenic, anti-inflammatory, and angiogenic adipokine that circulates in oligomeric complexes including: low molecular weight (LMW) trimers, medium molecular weight (MMW) hexamers, and high

molecular weight (HMW) isoforms. The aim of this study was to determine whether there are changes in adiponectin multimers in pregnancy and as a function of maternal weight. Study design. In this cross-sectional study, plasma concentrations of total, HMW, MMW, and LMW adiponectin were determined in women included in three groups: (1) normal pregnant women of normal body mass index (BMI) (n=466), (2) overweight pregnant women (BMI 25; n=257), and (3) non-pregnant women of normal weight (n=40). Blood samples were collected once from each woman between 11 and 42 weeks of gestation. Plasma adiponectin multimer concentrations were determined by enzyme-linked immunosorbent assay (ELISA). Non-parametric statistics were used for analysis. Results. (1) The median HMW adiponectin concentration and the median HMW/total adiponectin ratio were significantly higher, and the median LMW adiponectin concentration was significantly lower in pregnant women than in non-pregnant women. (2) Among pregnant women, the median plasma concentration of total, HMW, and MMW adiponectin was significantly higher in normal weight women than in overweight patients. (3) Maternal HMW was the most prevalent adiponectin multimer regardless of gestational age or BMI status. (4) There were no significant differences in the median concentration of total, MMW, and LMW adiponectin and their relative distribution with advancing gestation. Conclusion. Human pregnancy is characterized by quantitative and qualitative changes in adiponectin multimers, especially the most active isoform, HMW adiponectin.

3/7/58 (Item 6 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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18234265 Genuine Article#: 343IK Number of References: 26
Title: Blockade of serotonin 2A receptor improves glomerular endothelial function in rats with streptozotocin-induced diabetic nephropathy
Author(s): Kobayashi S; Satoh M (REPRINT) ; Namikoshi T; Haruna Y; Fujimoto S; Arakawa S; Komai N; Tomita N; Sasaki T; Kashiwara N
Corporate Source: Kawasaki Med Univ, Dept Internal Med, Div Nephrol, 577 Matsushima/Kurashiki/Okayama 7010192/Japan/ (REPRINT); Kawasaki Med Univ, Dept Internal Med, Div Nephrol, Kurashiki/Okayama 7010192/Japan/
Journal: CLINICAL AND EXPERIMENTAL NEPHROLOGY, 2008, V12, N2 (APR), P 119-125
ISSN: 1342-1751 Publication date: 20080400

Publisher: SPRINGER, 233 SPRING ST, NEW YORK, NY 10013 USA

Language: English Document Type: ARTICLE

Abstract: Background Serotonin (5-HT) is involved in vascular inflammation

and atherosclerogenesis. Serum 5-HT concentrations are elevated in diabetes, and 5-HT is involved in diabetic vasculopathies.

Sarpogrelate

hydrochloride, a 5-HT_{2A} receptor antagonist, has renoprotective effects, but its effect in diabetic nephropathy is not

elucidated. The

aim of this study was to examine the effects of sarpogrelate on endothelial dysfunction in rats with streptozotocin (STZ) induced diabetes.

Methods Rats with STZ-induced diabetes were either untreated or treated with sarpogrelate (30 mg/kg P.O.) for 8 weeks. At the end of the experiment, we measured urinary albumin excretion, serum

adiponectin concentration and platelet-derived microparticles. Intraglomerular coagulation was detected by immunostaining for

platelets. Production of renal reactive oxygen species (ROS) and nitric oxide (NO) was investigated by confocal laser microscopy and used as an

index of glomerular endothelial dysfunction.

Results Diabetic nephropathy was associated with enhanced production of ROS and diminished bioavailable NO in the glomeruli.

Treatment with sarpogrelate improved ROS/NO imbalance in glomeruli,

suppressed platelet aggregation in glomeruli, reduced platelet-derived microparticles, increased serum adiponectin level and reduced the level of albuminuria, compared with

nontreated

diabetic rats.

Conclusions Our results indicate that sarpogrelate improves endothelial function in rats with STZ-induced diabetes through a reduction of glomerular platelet activation and an increase in

serum

adiponectin concentrations and suggest that sarpogrelate is potentially useful for the treatment of diabetic nephropathy.

3/7/59 (Item 7 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

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17940703 Genuine Article#: 309IS Number of References: 34

Title: Adiponectin induces dentin sialophosphoprotein in rat dental pulp cells: An in vitro study

Author(s): Yasuda Y (REPRINT) ; Koike T; Kawamorita T; Saito T

Corporate Source: Hlth Sci Univ Hokkaido, Sch Dent, Dept Oral Rehabil,
Div

Clin Cariol & Endodontol, 1757 Kanazawa/Ishikari/Hokkaido
0610293/Japan/

(REPRINT); Hlth Sci Univ Hokkaido, Sch Dent, Dept Oral Rehabil,
Div Clin

Cariol & Endodontol, Hokkaido//Japan/

Journal: JOURNAL OF ENDODONTICS, 2008, V34, N6 (JUN), P679-683

ISSN: 0099-2399 Publication date: 20080600

Publisher: ELSEVIER SCIENCE INC, 360 PARK AVE SOUTH, NEW YORK, NY
10010-1710 USA

Language: English Document Type: ARTICLE

Abstract: Adiponectin is known to play an important role in the
regulation of blood glucose levels through the mediation of
adiponectin receptors 1 and 2 (AR1 and AR2, respectively). The
purpose of this study was to investigate the role of adiponectin
in dental pulp cells. The expressions of both AR1 and AR2 were
observed

in dental pulp by reverse transcriptase polymerase chain reaction
(RT-PCR) and Western blotting. Quantitative analysis of Alizarin

Red S

staining showed that 10 μ g/mL of adiponectin significantly
promoted mineralization by 1.6 times compared with control on day

12.

However, no significant difference in mineralization was observed
between control and 0.1 or 1 μ g/mL adiponectin treatment.

Moreover, real-time PCR results indicated that adiponectin (10 μ
g/mL) significantly increased the expression of dentin
sialophosphoprotein (DSPP) by 2.3 and 1.8 times compared with

control

on days 8 and 12, respectively. These results indicated that
adiponectin might promote mineralization by inducing DSPP
expression in dental pulp cells.

3/7/60 (Item 8 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

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17846964 Genuine Article#: 302BQ Number of References: 30

Title: Globular adiponectin induces platelet activation through the
collagen receptor GPVI-Fc receptor gamma chain complex

Author(s): Riba R; Hughes CE; Graham A; Watson SP; Naseem KM
(REPRINT)

Corporate Source: Univ Bradford, Ctr Atherothrombosis Res, Bradford BD7
1DP/W

Yorkshire/England/ (REPRINT); Univ Bradford, Ctr Atherothrombosis
Res, Bradford BD7 1DP/W Yorkshire/England/; Inst Biomed Res, Ctr
Cardiovasc Sci, Birmingham//AL/; Glasgow Caledonian Univ, Vascular

Biol

Grp Biol & Biomed Sci, Glasgow G4 0BA/Lanark/Scotland/

Journal: JOURNAL OF THROMBOSIS AND HAEMOSTASIS, 2008, V6, N6 (JUN), P

1012-1020

ISSN: 1538-7933 Publication date: 20080600

Publisher: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ,
OXON,

ENGLAND

Language: English Document Type: ARTICLE

Abstract: Background: The adipocyte-derived cytokine, adiponectin
(Ad), exerts potent vascular effects, although the direct effects
of Ad

on blood platelets are unclear. Objective: The influence of
globular Ad

(gAd) on blood platelet function was investigated. Research
design and

methods: We measured platelet aggregation and tyrosine
phosphorylation signaling events in human and mouse platelets. The
ability of gAd to activate Glycoprotein VI (GPVI) activity was
determined with a NFAT luciferase reporter assay. Results: gAd,
but not

full length Ad, induced rapid aggregation and granule secretion
of human and mouse platelets through a pathway that is ablated
under

conditions of Src kinase inhibition, indicating a tyrosine
kinase-dependent mechanism. Consistent with this, gAd stimulates
rapid

tyrosine phosphorylation of several proteins in human and mouse
platelets. The pattern of increase in tyrosine phosphorylation was
similar to that induced by collagen, with the tyrosine kinase Syk
and

PLC gamma 2 being identified among the list of tyrosine
phosphorylated

proteins. As collagen activates platelet through the GPVI-Fc
receptor

gamma-chain (FcR gamma) complex, we used FcR gamma null platelets
(which also lack GPVI) to explore the mechanism by which gAd
stimulates

platelets. Stimulation of tyrosine phosphorylation and platelet
aggregation by gAd was abolished in FcR gamma null platelets and
markedly reduced in the absence of PLC gamma 2. Further, GPVI was
confirmed as a collagen receptor for gAd by increased luciferase
activity in Jurkat T-cells transfected with GPVI. Conclusions: We
identify gAd as a novel ligand for GPVI that stimulates tyrosine
kinase-dependent platelet aggregation. Our data raise the
possibility that gAd may promote unwanted platelet activation at
sites

of vascular injury.

3/7/61 (Item 9 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

(c) 2009 The Thomson Corp. All rts. reserv.

17679944 Genuine Article#: 283HX Number of References: 49

Title: Adiponectin: An adipocytic hormone implicated in carbohydrate homeostasis and cardiovascular function

Author(s): Guerre-Millo M (REPRINT)

Corporate Source: Hop Hotel Dieu, AP HP, INSERM, Serv Nutr, U 755, 1 Pl Parvis

Notre Dame/F-75004 Paris//France/ (REPRINT); Hop Hotel Dieu, AP HP, INSERM, Serv Nutr, U 755, F-75004 Paris//France/

Journal: SANG THROMBOSE VAISSEAUX, 2007, V19, N5 (MAY), P255-260

ISSN: 0999-7385 Publication date: 20070500

Publisher: JOHN LIBBEY EUROTTEXT LTD, 127 AVE DE LA REPUBLIQUE, 92120 MONTRouGE, FRANCE

Language: French Document Type: REVIEW

Abstract: The discovery of leptin and adiponectin, two proteins produced by adipose cells and released in the circulation, is considered as a breakthrough in the field of metabolic diseases.

Leptin

has been clearly characterized as a hormone, which modulates food intake depending on energy status. By contrast, a biological role for

adiponectin has not yet been firmly established. Nevertheless, numerous clinical and experimental observations indicate that low adiponectin plasma levels contribute to the pathogenesis of insulin resistance, type 2 diabetes and cardiovascular diseases in obese or overweight patients. Indeed, adiponectin exerts both antiatherogenic effects, by targeting vascular endothelial cells, and

insulin-sensitizing effects, prominently in muscles and liver.

Among

several circulating forms, a high molecular weight multimer of adiponectin is thought to be the most clinically relevant. Adiponectin signalling pathways comprise at least two putative receptors, AdipoR1 and AdipoR2, which mediate increased fatty acid oxidation in muscles and fat, decreased glucose production in liver and

reduced inflammation-related processes in endothelial cells, at least

in part through activation of AMP kinase. Methods of improving adiponectin bioactivity, are under intense investigation. This includes the use of drugs such as thiazolidinediones and Cb1 blockers

(rimonabant), which increase adiponectin gene expression and plasma levels. Alternatively, the development of AdipoR agonists could

prove beneficial in situations such as obesity, where decreased serum

adiponectin levels are observed.

3/7/62 (Item 10 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

(c) 2009 The Thomson Corp. All rts. reserv.

17582579 Genuine Article#: 276LN Number of References: 40
Title: Genetic and environmental determinants of hepatocyte growth factor

levels and their association with obesity and blood pressure
Author(s): Vistoropsky Y; Trofimov S; Malkin I; Kobylansky E; Livshits G

(REPRINT)

Corporate Source: Tel Aviv Univ, Sackler Fac Med, Dept Anat & Anthropol, Yoran Inst Human Genome Res, Human Populat Biol Res Unit, IL-69978 Tel Aviv//Israel/ (REPRINT); Tel Aviv Univ, Sackler Fac

Med, Dept Anat & Anthropol, Yoran Inst Human Genome Res, Human Populat

Biol Res Unit, IL-69978 Tel Aviv//Israel/

Journal: ANNALS OF HUMAN BIOLOGY, 2008, V35, N1 (JAN-FEB), P93-103

ISSN: 0301-4460 Publication date: 20080100

Publisher: TAYLOR & FRANCIS LTD, 4 PARK SQUARE, MILTON PARK, ABINGDON OX14

4RN, OXON, ENGLAND

Language: English Document Type: ARTICLE

Abstract: Background: Hepatocyte growth factor (HGF) is a member of the

adipocytokine family; it is implicated in tissue repair, regeneration,

and angiogenesis. Several studies have reported that the HGF plays important role in obesity and cardiovascular disease.

Aim: This study examines whether HGF and its phenotypic correlations with obesity and blood pressure (BP), in healthy individuals, are due to shared genetic or common environmental factors.

Subjects and methods: Body mass index (BMI), waist-to-hip ratio

(WHR), BP, and HGF plasma concentrations were measured in a sample of

733 individuals belonging to 248 pedigrees.

Results: The most significant phenotypic correlations were found

among HGF, WHR, and systolic BP ($p < 0.001$). Analysis of the familial

aggregation revealed that parent-offspring and sibling correlations in HGF levels, adjusted for age, age², and sex, were statistically highly significant ($p < 0.001$). Variance decomposition

analysis showed that when adjusted for potential covariates, 48.4% of

the HGF variation was due to putative genetic factors. The genetic correlations between all pairs of studied traits (HGF, WHR, and SBP)

were statistically significant ($p < 0.02$) and ranged between 0.23 +/-

0.07 and 0.40 +/- 0.07. However, correlation between WHR and BP becomes non-significant after adjustment for HGF.

Conclusions: The results provide evidence that putative genetic factors involved in regulation of HGF variation contribute also significantly to variation of the obesity and BP. It is possible that the familial resemblance for WHR and the SBP correlation in the studied sample is affected substantially by genetic factors regulating circulating HGF levels.

3/7/63 (Item 11 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

17440645 Genuine Article#: 256JY Number of References: 0
Title: 5-HT2 receptor blockade improves cerebral infarction in diabetic rodent models by inducing adiponectin expression and inhibiting platelet aggregation
Author(s): Nakagawa H; Uchida S; Yamada K; Shimada H; Akira T; Kitada Y
Corporate Source: Mitsubishi Pharma Corp, Yokohama/Kanagawa/Japan/
Journal: STROKE, 2008, V39, N2 (FEB), P658-658
ISSN: 0039-2499 Publication date: 20080200
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA
Language: English Document Type: MEETING ABSTRACT

3/7/64 (Item 12 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

17088144 Genuine Article#: 2280D Number of References: 31
Title: A low level of C-reactive protein in Japanese adults and its association with cardiovascular risk factors: The Japan NCVC-Collaborative Inflammation Cohort (JNIC) Study
Author(s): Saito I (REPRINT) ; Sato S; Nakamura M; Kokubo Y; Mannami T; Adachi H; Konishi M; Okada K; Iso H; Kario K; Ohsuzu F; Momiyama Y; Tsushima M
Corporate Source: Ehime Univ, Grad Sch Med, Dept Publ Hlth Social Med & Med Informat, Toon/Ehime 7910295/Japan/ (REPRINT); Ehime Univ, Grad Sch Med,

Dept Publ Hlth Social Med & Med Informat, Toon/Ehime
7910295/Japan/;

Nara Med Univ, Dept Publ Hlth Policy, Kashihara/Nara 634/Japan/;
Osaka

Med Ctr Hlth Sci & Promot, Osaka//Japan/; Natl Cardiovasc Ctr, Dept
Prevent Cardiol, Suita/Osaka 565/Japan/; Kagawa Univ, Fac Med, Dept
Hyg

Publ Hlth, Kagawa//Japan/; Kurume Univ, Sch Med, Dept Internal Med,
Div

Cardiovasc Med, Kurume/Fukuoka 830/Japan/; Osaka Univ, Grad Sch
Med, Dept

Social & Environm Med, Suita/Osaka/Japan/; Jichi Med Sch, Dept
Cardiol, Tochigi//Japan/; Natl Def Med Coll, Dept Internal Med
1, Saitama//Japan/; Int Univ Hlth & Welfare, Atami Hosp, Dept
Geriatr

Med, Atami//Japan/

Journal: ATHEROSCLEROSIS, 2007, V194, N1 (SEP), P238-244

ISSN: 0021-9150 Publication date: 20070900

Publisher: ELSEVIER IRELAND LTD, ELSEVIER HOUSE, BROOKVALE PLAZA,
EAST PARK

SHANNON, CO, CLARE, 00000, IRELAND

Language: English Document Type: ARTICLE

Abstract: High-sensitivity C-reactive protein (hs-CRP) levels vary
remarkably by race and ethnic group. We examined hs-CRP levels and
their association with cardiovascular risk factors in the Japanese
general population. The Japan National Cardiovascular Center
(NCVC)-collaborative Inflammation Cohort (JNIC) Study recruited
5213

men and 7071 women aged ≥ 40 years from seven communities in
Japan

during 2002-2004. hs-CRP was measured using nephelometry
calibrated

with CRM 470, the international plasma protein reference material.
Traditional cardiovascular risk factors and their aggregation
were studied in multivariate logistic models, stratified by
overweight

status. Median hs-CRP levels in men and women were 0.60 and 0.45
mg/L,

respectively. The percentage of subjects with hs-CRP levels < 1.0 ,
1.0-3.0, and > 3.0 mg/L was 67.4%, 22.0%, and 10.6% in men,
respectively, and 76.3%, 16.7%, and 7.0% in women. hs-CRP levels
showed

significant linear associations with traditional risk factors.

Overweight, hypertension, dyslipidemia (men only), smoking (men
only),

and diabetes (women only) contributed significantly to elevated
hs-CRP

levels. Overweight individuals with hypertension, dyslipidemia,
and

diabetes had a high prevalence of elevated hs-CRP levels in both
sexes.

Japanese adults have very low hs-CRP levels. An aggregation of

metabolic risk factors is associated with elevated hs-CRP levels among overweight individuals, particularly in women. (C) 2006 Elsevier Ireland Ltd. All rights reserved.

3/7/65 (Item 13 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

17064228 Genuine Article#: 225TC Number of References: 81
Title: Genetic factors in diabetic nephropathy
Author(s): Freedman BI (REPRINT) ; Bostrom M; Daeihagh P; Bowden DW
Corporate Source: Wake Forest Univ,Sch Med, Nephrol Sect, Dept Internal
Med,Med Ctr Blvd/Winston Salem//NC/27157 (REPRINT); Wake Forest Univ,Sch Med, Nephrol Sect, Dept Internal Med,Winston Salem//NC/27157;
Wake Forest Univ,Sch Med, Dept Biochem,Winston Salem//NC/27109;
Wake Forest Univ,Sch Med, Ctr Human Genom,Winston Salem//NC/27109
Journal: CLINICAL JOURNAL OF THE AMERICAN SOCIETY OF NEPHROLOGY, 2007, V2, N6 (NOV), P1306-1316
ISSN: 1555-905X Publication date: 20071100
Publisher: AMER SOC NEPHROLOGY, 1725 I ST, NW STE 510, WASHINGTON, DC 20006

USA
Language: English Document Type: REVIEW
Abstract: Several genes that predispose to type 2 diabetes have recently been identified. In addition to the recognized and powerful effects of environmental factors, there is abundant evidence in support of genetic susceptibility to the microvascular complication of nephropathy in individuals with both type 1 and type 2 diabetes. Familial aggregation of phenotypes such as end-stage renal disease, albuminuria, and chronic kidney disease have routinely been reported in populations throughout the world, and heritability estimates for albuminuria and glomerular filtration rate demonstrate strong contributions of inherited factors. Recent genome-wide linkage scans have identified several chromosomal regions that likely contain diabetic nephropathy susceptibility genes, and association analyses have evaluated positional candidate genes under these linkage peaks. These complimentary approaches have demonstrated that polymorphisms in the carnosinase 1 gene on chromosome 18q, the adiponectin gene on

3q, and the engulfment and cell motility gene on 7p are likely associated with susceptibility to diabetic nephropathy. Additional genes that seem to be of importance in renal phenotypes include manganese superoxide dismutase and angiotensin 1-converting enzyme, with nitric oxide synthase implicated in albuminuria. This article reviews the inherited aspects of diabetic kidney disease with particular emphasis on recently implicated genes and pathways. It seems likely that the risk for diabetes-associated kidney disease is magnified by inheriting risk alleles at several susceptibility loci, in the presence of hyperglycemia.

3/7/66 (Item 14 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

16967537 Genuine Article#: 213PM Number of References: 144
Title: Strategies to reduce vascular risk associated with obesity
Author(s): Bodary PF (REPRINT) ; Lglay HB; Eitzman DT
Corporate Source: 410 W Warren Ave,3009 Sci Hall/Detroit//MI/48202
(REPRINT); Wayne State Univ,Coll Liberal Arts & Sci, Dept Nutr & Food
Sci,Detroit//MI/48202; Univ Michigan,Dept Internal Med, Div Cardiovasc
Med,Ann Arbor//MI/48109
Journal: CURRENT VASCULAR PHARMACOLOGY, 2007, V5, N4 (OCT), P249-258
ISSN: 1570-1611 Publication date: 20071000
Publisher: BENTHAM SCIENCE PUBL LTD, EXECUTIVE STE Y26, PO BOX 7917, SAIF
ZONE, 1200 BR SHARJAH, U ARAB EMIRATES
Language: English Document Type: REVIEW
Abstract: The obesity pandemic will likely have a significant impact on the
global incidence of cardiovascular disease. Although the mechanisms
linking obesity and cardiovascular disease are unclear, recent studies
have implicated the adipocyte as a potentially important mediator of
vascular complications. The adipocyte is no longer considered a passive
storage depot for triglycerides and fatty acids, but rather an active
metabolic organ capable of producing several factors, commonly referred
to as adipokines, that may have effects on many physiological and pathophysiological processes. With increasing fat mass, several adipose-related factors are upregulated that may affect local and distant inflammatory processes, including atherothrombosis. Other

factors, such as adiponectin, are downregulated with increasing fat mass. Although most adipokines are thought to promote vascular disease, several studies over the past few years indicate adiponectin is actually protective against both diabetes and vascular disease. There are now available pharmacologic agents capable of altering the adipocyte transcription profile. This review will focus on the potential impact of adipocyte-derived factors towards vascular disease and emerging therapeutic strategies that may alter these effects.

3/7/67 (Item 15 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

16808853 Genuine Article#: 206KE Number of References: 21
Title: Low molecular weight adiponectin negatively correlates with the waist circumference and monocytic IL-6 release
Author(s): Schober F; Neumeler M; Weigert J; Wurm S; Wanninger J; Schaffler A; Dada A; Liebisch G; Schmitz G; Aslanidis C; Buechler C (REPRINT)
Corporate Source: Regensburg Univ Hosp, Dept Internal Med 1, D-93042 Regensburg//Germany/ (REPRINT); Regensburg Univ Hosp, Dept Internal Med 1, D-93042 Regensburg//Germany//; Regensburg Univ Hosp, Inst Clin Chem, D-93042 Regensburg//Germany//; Regensburg Univ Hosp, Lab Med, D-93042 Regensburg//Germany/
Journal: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, 2007, V361, N4 (OCT 5), P968-973
ISSN: 0006-291X Publication date: 20071005
Publisher: ACADEMIC PRESS INC ELSEVIER SCIENCE, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495 USA
Language: English Document Type: ARTICLE
Abstract: Adiponectin circulates as trimer (LMW), hexamer (MMW) and high molecular weight multimer (HMW) but the distribution and effects of these isoforms have not been studied in detail. Monocytes were isolated from normal weight and overweight controls and patients with type 2 diabetes mellitus (T2D) and monocytic release of IL-6 positively correlated with the body mass index (BMI). HMW-adiponectin further enhanced and LMW-adiponectin reduced IL-6 release in monocytes. Systemic total adiponectin, and the HMW isoform were not different in these groups but MMW-adiponectin was lower in T2D, and LMW-adiponectin was

reduced in the obese and T2D. Circulating LMW-adiponectin negatively correlated to monocytic IL-6 release. Systemic IL-6 was higher in the obese control group and T2D, respectively, but did not correlate with monocytic IL-6 secretion. Therefore, the current study indicates that HMW-adiponectin exerts pro- and LMW-adiponectin antiinflammatory effects and reduced LMW-adiponectin in obesity may partly contribute to elevated monocytic IL-6 release. (C) 2007 Elsevier Inc. All rights reserved.

3/7/68 (Item 16 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

16658303 Genuine Article#: 183XL Number of References: 123
Title: Inflammatory responses underlying the microvascular dysfunction associated with obesity and insulin resistance
Author(s): Singer G; Granger DN (REPRINT)
Corporate Source: Louisiana State Univ,Hlth Sci Ctr, Dept Mol & Cellular Physiol,1501 Kings Highway/Shreveport//LA/71130 (REPRINT); Louisiana State Univ,Hlth Sci Ctr, Dept Mol & Cellular Physiol,Shreveport//LA/71130; Med Univ Graz,Dept Pediat Surg,Graz//Austria/
Journal: MICROCIRCULATION, 2007, V14, N4-5, P375-387
ISSN: 1073-9688 Publication date: 20070000
Publisher: TAYLOR & FRANCIS INC, 325 CHESTNUT ST, SUITE 800, PHILADELPHIA, PA 19106 USA
Language: English Document Type: REVIEW
Abstract: Obesity is a growing health care problem that is increasing the incidence and morbidity of cardiovascular diseases. Emerging evidence suggests that obesity is associated with a systemic inflammatory response that is characterized by endothelial cell dysfunction, oxidative stress, and the activation of circulating immune cells. Adipocytes produce and release a variety of cytokines (IL-1, TNF-alpha) and cytokine-like substances (leptin, resistin) that appear to mediate the inflammatory response that accompanies obesity. The abrogating influence of weight loss on the inflammatory response supports this contention. The insulin resistance that often accompanies obesity may also contribute to this inflammatory phenotype. Studies in experimental

animals and clinical studies suggest that the microvascular dysfunction associated with pathological states, such as sepsis, is greatly exacerbated by obesity. Although the microvasculature appears to be a major target for the deleterious inflammatory consequences of obesity, relatively little attention has been devoted to characterizing the effects of obesity on inflammatory responses in different regional vascular beds and to defining the mechanisms that underlie the resultant microvascular dysfunction.

3/7/69 (Item 17 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

16559232 Genuine Article#: 172TU Number of References: 97
Title: Adipokines and coronary vasomotor dysfunction
Author(s): Knudson JD; Dick GM; Tune JD (REPRINT)
Corporate Source: Indiana Univ, Sch Med, Dept Cellular & Integrat
Physiol, 635 Barnhill Dr/Indianapolis//IN/46202 (REPRINT); Indiana
Univ, Sch Med, Dept Cellular & Integrat
Physiol, Indianapolis//IN/46202;
Louisiana State Univ, Hlth Sci Ctr, Dept Physiol, New
Orleans//LA/70112
Journal: EXPERIMENTAL BIOLOGY AND MEDICINE, 2007, V232, N6 (JUN),
P727-736
ISSN: 1535-3702 Publication date: 20070600
Publisher: SOC EXPERIMENTAL BIOLOGY MEDICINE, 195 WEST SPRING VALLEY
AVE,
MAYWOOD, NJ 07607-1727 USA
Language: English Document Type: REVIEW
Abstract: Research in the last 10-15 years has shown that fat cells
(adipocytes) produce and release proteins with specific biologic
activities. These proteins, termed adipokines, include the
hormones
leptin, adiponectin, and resistin. Adipose tissue is now
recognized as an active endocrine organ. With the obesity pandemic
swelling in the Western world, ongoing research is aimed at
determining
the biologic links between obesity and cardiovascular disease.
This
review presents basic historical background information on the
major
adipokines, introduces findings from clinical studies associating
adipokines with cardiovascular disease, and summarizes results
from
recent basic science research studies of mechanisms of
adipokine-induced cardiovascular dysfunction. Particular emphasis
is
placed on the action of adipokines in the coronary

circulation-especially effects of adipokines on endothelial function,
as endothelial damage is likely a critical event initiating atherosclerotic coronary artery disease.

3/7/70 (Item 18 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

15150084 Genuine Article#: 041GJ Number of References: 48
Title: The role of adiponectin in atherosclerosis and thrombosis
Author(s): Ekmekci H (REPRINT) ; Ekmekci OB
Corporate Source: Miralay Hasan Kazim Sok, Ersevenler Sit, Mehtap
Apt/TR-34290 Istanbul//Turkey/ (REPRINT); Univ Istanbul, Fac Med,
Dept
Pediat Haematol & Oncol, Istanbul//Turkey/; Univ Istanbul, Bone
Marrow
Transplantat Unit, Cerrahpasa Med Fac, Dept
Biochem, Istanbul//Turkey/(
hakekmekci@yahoo.com)
Journal: CLINICAL AND APPLIED THROMBOSIS-HEMOSTASIS, 2006, V12, N2
(APR), P
163-168
ISSN: 1076-0296 Publication date: 20060400
Publisher: WESTMINSTER PUBL INC, 708 GLEN COVE AVE, GLEN HEAD, NY
11545 USA
Language: English Document Type: ARTICLE
Abstract: Obesity is a major risk factor for morbidity and mortality
from
cardiovascular causes. Adiponectin has been identified recently
as one of the adipocytokines with important metabolic effects. it
can
suppress atherogenesis by inhibiting the adherence of monocytes,
reducing their phagocytic activity, and suppressing the
accumulation of
modified lipoproteins in the vascular wall. In addition, as
adiponectin decrease endothelial damage and stimulates production
of NO from vascular endothelial cells, hypoadiponectinemia may be
partially contribute to thrombus formation.

3/7/71 (Item 19 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

14437979 Genuine Article#: 971NH Number of References: 118
Title: Treating insulin resistance in type 2 diabetes with metformin
and
thiazolidinediones
Author(s): Bailey CJ (REPRINT)
Corporate Source: Aston Univ, Sch Life & Hlth Sci, Birmingham B4 7ET/W

Midlands/England/ (REPRINT); Aston Univ,Sch Life & Hlth
Sci,Birmingham

B4 7ET/W Midlands/England/(c.j.bailey@aston.ac.uk)

Journal: DIABETES OBESITY & METABOLISM, 2005, V7, N6 (NOV), P675-691

ISSN: 1462-8902 Publication date: 20051100

Publisher: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ,
OXON,

ENGLAND

Language: English Document Type: REVIEW

Abstract: Insulin resistance underlies the pathogenesis of
hyperglycaemia

and cardiovascular disease in most people with type 2 diabetes.

Metformin and thiazolidinediones (pioglitazone and rosiglitazone)
counter insulin resistance by different cellular mechanisms and

with

complementary effects, making them suited for use in combination.
Metformin exerts a stronger suppression of hepatic glucose output,
while thiazolidinediones produce a greater increase in peripheral
glucose uptake, enabling metformin-thiazolidinedione combinations

to

improve glycaemic control in type 2 diabetes with additive
efficacy.

Basal insulin concentrations are not raised by metformin or
thiazolidinediones, so there is minimal risk of hypoglycaemia, and
metformin can reduce the weight gain associated with
thiazolidinediones. There are overlapping effects of metformin and
thiazolidinediones against a range of athero-thrombotic factors

and

markers. These include decreased plasminogen activator
inhibitor-1,

reduced platelet aggregation, reductions of several vascular
adhesion molecules, and reduced markers of low-grade inflammation

such

as C-reactive protein. Additionally, thiazolidinediones increase
adiponectin and slightly reduce blood pressure. Both metformin
and thiazolidinediones can improve components of the lipid

profile:

thiazolidinediones consistently reduce free fatty acid
concentrations

and decrease the proportion of small dense
low-density-lipoprotein, and

pioglitazone also decreases triglycerides. During
co-administration,

metformin and thiazolidinediones do not interfere with each
other's

pharmacokinetics, and lower doses of the two agents together can
achieve efficacy with fewer side effects.

Metformin-thiazolidinedione

combinations require attention to the precautions for both agents,
especially renal, cardiac and hepatic status. Thus, metformin and
thiazolidinediones can be used in combination to address the
hyperglycaemia and vascular risk in type 2 diabetes. .

3/7/72 (Item 20 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

14332480 Genuine Article#: 961LZ Number of References: 38
Title: Mouse and human resistins impair glucose transport in primary mouse

cardiomyocytes, and oligomerization is required for this
biological
action

Author(s): Gravelleau C; Zaha VG; Mohajer A; Banerjee RR;
Dudley-Rucker N;

Steppan CM; Rajala MW; Scherer PE; Ahima RS; Lazar MA; Abel ED
(REPRINT)

Corporate Source: Univ Utah, Sch Med, Div Endocrinol Diabet & Metab, 15
North

2030 East, Bldg 533, Rm 3410B/Salt Lake City//UT/84112 (REPRINT);
Univ

Utah, Sch Med, Div Endocrinol Diabet & Metab, Salt Lake
City//UT/84112;

Univ Utah, Program Human Mol Biol & Genet, Salt Lake
City//UT/84112; Univ

Penn, Sch Med, Div Endocrinol Diabet &
Metab, Philadelphia//PA/19104;

Yeshiva Univ Albert Einstein Coll Med, Dept Cell
Biol, Bronx//NY/10461(
dale.abel@hmbg.utah.edu)

Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 2005, V280, N36 (SEP 9), P
31679-31685

ISSN: 0021-9258 Publication date: 20050909

Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE
PIKE, BETHESDA, MD 20814-3996 USA

Language: English Document Type: ARTICLE

Abstract: The adipocytokine resistin impairs glucose tolerance and
insulin

sensitivity in rodents. Here, we examined the effect of resistin
on

glucose uptake in isolated adult mouse cardiomyocytes. Murine
resistin

reduced insulin-stimulated glucose uptake, establishing the heart
as a

resistin target tissue. Notably, human resistin also impaired
insulin

action in mouse cardiomyocytes, providing the first evidence that
human

and mouse resistin homologs have similar functions. Resistin is a
cysteine-rich molecule that circulates as a multimer of a dimeric
form dependent upon a single intermolecular disulfide bond,
which, in

the mouse, involves Cys(26); mutation of this residue to alanine
(C26A)

produces a monomeric molecule that appears to be bioactive in the liver. Remarkably, unlike native resistin, monomeric C26A resistin had no effect on basal or insulin-stimulated glucose uptake in mouse cardiomyocytes. Resistin impairs glucose uptake in cardiomyocytes by mechanisms that involve altered vesicle trafficking. Thus, in cardiomyocytes, both mouse and human resistins directly impair glucose transport; and in contrast to effects on the liver, these actions of resistin require oligomerization.

3/7/73 (Item 1 from file: 45)
DIALOG(R)File 45:EMCare
(c) 2009 Elsevier B.V. All rts. reserv.

0005540660 EMCARE No: 354470016
Exendin-4 improves glycemic control, ameliorates brain and pancreatic pathologies, and extends survival in a mouse model of huntington's disease
Martin B.; Golden E.; Carlson O.D.; Pistell P.; Zhou J.; Kim W.; Frank B.P.; Thomas S.; Chadwick W.A.; Greig N.H.; Bates G.P.; Sathasivam K.; Bernier M.; Maudsley S.; Mattson M.P.; Egan J.M.
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OBJECTIVE-The aim of this study was to find an effective treatment for the genetic form of diabetes that is present in some Huntington's disease patients and in Huntington's disease mouse models. Huntington's disease is a neurodegenerative disorder caused by a polyglutamine expansion within the

huntingtin protein. Huntington's disease patients exhibit neuronal dysfunction/degeneration, chorea, and progressive weight loss. Additionally, they suffer from abnormalities in energy metabolism affecting both the brain and periphery. Similarly to Huntington's disease patients, mice expressing the mutated human huntingtin protein also exhibit neurodegenerative changes, motor dysfunction, perturbed energy metabolism, and elevated blood glucose levels. RESEARCH DESIGN AND METHODS-Huntington's disease mice were treated with an FDA-approved antidiabetic glucagon-like peptide 1 receptor agonist, exendin-4 (Ex-4), to test whether euglycemia could be achieved, whether pancreatic dysfunction could be alleviated, and whether the mice showed any neurological benefit. Blood glucose and insulin levels and various appetite hormone concentrations were measured during the study. Additionally, motor performance and life span were quantified and mutant huntingtin (mhtt) aggregates were measured in both the pancreas and brain. RESULTS-Ex-4 treatment ameliorated abnormalities in peripheral glucose regulation and suppressed cellular pathology in both brain and pancreas in a mouse model of Huntington's disease. The treatment also improved motor function and extended the survival time of the Huntington's disease mice. These clinical improvements were correlated with reduced accumulation of mhtt protein aggregates in both islet and brain cells. CONCLUSIONS-Targeting both peripheral and neuronal deficits, Ex-4 is an attractive agent for therapeutic intervention in Huntington's disease patients suffering from diabetes. (c) 2009 by the American Diabetes Association.

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Endocrine Functions of Adipose Tissue: Focus on Adiponectin
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Accumulating evidence indicates that obesity and overweight are associated with, and contribute to, the development of type 2 diabetes mellitus (DM), cardiovascular disease (CVD), and chronic kidney disease

(CKD). The adipocyte-derived cytokine, adiponectin, has been shown to improve insulin sensitivity, increase rates of fatty acid oxidation, decrease muscle lipid content, and reduce inflammation and vascular injury.

However, adiponectin levels have been found to be reduced in persons with obesity and type 2 DM. Furthermore, adiponectin levels are inversely associated with those of tumor necrosis factor- α and C-reactive protein-markers of endothelial dysfunction and systemic inflammation. The 2 receptors for adiponectin-Adipo R SUB 1 and Adipo R SUB 2 , which are expressed in muscle and liver tissue and in human fat

cells-are hormonally regulated, with increased insulin levels causing a

reduction in their abundance. The hyperinsulinemia observed in obesity,

therefore, may be partially responsible for the reduction in the numbers of

adiponectin receptors. Adiponectin aggregates range from a hexamer of low molecular weight to larger multimeric structures of high

molecular weight. A smaller proteolytic fragment-the globular head domain

of adiponectin, or gAd-interacts specifically with skeletal muscle.

The relation of circulating adiponectin to its biologic actions is more complex than originally believed; therefore, it is the multimeric forms of the adiponectin molecule that need to be measured and

evaluated in relation to associated metabolic, cardiovascular, and renal functions. Furthermore, strategies to measure the numbers of adiponectin receptors on available tissue need to be developed to fully assess the clinical role of adiponectin in type 2 DM, CVD, and CKD. (c) 2008 Excerpta Medica.

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DIALOG(R)File 45:EMCare
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0005326099 EMCARE No: 351486145
Alcohol consumption and heart failure: A systematic review
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NUMBER OF REFERENCES: 45

Heart failure (HF) remains a major public health issue. It is estimated that about 500,000 Americans per year are diagnosed with HF. Despite advanced medical and surgical treatments for HF, mortality after the onset of HF is still high, thereby underscoring the importance of primary prevention. Among modifiable lifestyle factors, alcohol consumption appears to play a role in the development of HF. Although excessive drinking has been known to lead to alcoholic cardiomyopathy and light-to-moderate drinking may confer some cardiovascular benefits, recent studies suggest it is not only the quantity, but also drinking patterns and genetic factors, that may influence the relation between alcohol consumption and cardiovascular disease. This article reviews current evidence on the

association between alcohol consumption and HF. Copyright (c) 2008 by Current Medicine Group LLC.

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Metabolic syndrome: An appraisal of the pro-inflammatory and procoagulant status

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Inflammation and hypercoagulability predispose to atherothrombosis and

seem to be important features of the metabolic syndrome. The most convincing evidence is the association with increased levels of C-reactive

protein. The hemostatic abnormality that has been most consistently associated with insulin resistance is an elevated plasminogen activator

inhibitor-1 level. In contrast, markers of hypercoagulability have been

associated inconsistently with hyperinsulinemia and glucose intolerance.

Fibrinogen clusters with inflammatory factors, which suggests involvement

of adipose tissue-generated inflammatory cytokines. Elevated von Willebrand's factor and factor VIII levels aggregate with indicators of endothelial injury, whereas vitamin K-dependent coagulation proteins

correlate with triglyceride levels.

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DIALOG(R)File 65:Inside Conferences
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05134031 INSIDE CONFERENCE ITEM ID: CN053441735
Enhanced Platelet Aggregation and Thrombogenic Tendency in
Adiponectin-Deficient Mice. (8:15 AM)
Kato, H.; Kashiwagi, H.; Shiraga, M.; Honda, S.; Miyata, S.;
Yamamoto,
J.; Kurata, Y.; Funahashi, T.; Shimomura, I.; Tomiyama, Y.
CONFERENCE: American Society of Hematology; Abstracts for the 46th
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Anti-inflammatory and cardioprotective effects of n-3 polyunsaturated
fatty
acids and plant sterols in hyperlipidemic individuals
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Background: Risk factors of cardiovascular disease such as lipid aberrations, hypertension, abdominal adiposity and elevations in systemic inflammation, are prominent aetiologies in hyperlipidemia. Supplementation with n-3 PUFA is associated with a reduction in cardiovascular events through its hypotriglyceridemic, anti-aggregatory and anti-inflammatory properties. Plant sterols have potent hypocholesterolemic properties, although their effect on the inflammatory cascade is uncertain. This study investigated the effect of combined supplementation with n-3 PUFA and plant sterols on cardiovascular risk factors, blood pressure, body composition, markers of systemic inflammation and overall risk, in hyperlipidemic individuals. Methods: The study was a 3-week randomised, double-blind, placebo-controlled, 2 x 2 factorial design, in four parallel groups. Sixty hyperlipidemic participants were randomised to receive either sunola oil or 1.4 g/d n-3 PUFA capsules with or without 2 g plant sterols per day. Results: The combination of n-3 PUFA and plant sterols reduced several inflammatory markers. High sensitivity C-reactive protein (hs-CRP) was reduced by 39% (P = 0.009), tumor necrosis factor-alpha (TNF-alpha) by 10% (P = 0.02), interleukin-6 (IL-6) by 10.7% (P = 0.009), leukotriene B₄ (LTB₄) by 29.5% (P = 0.01) and adiponectin was increased by 29.5% (P = 0.05). Overall cardiovascular risk was reduced by 22.6% (P = 0.006) in the combination group. Conclusion: We have demonstrated, for the first time that dietary intervention with n-3 PUFA and plant sterols reduces systemic inflammation in hyperlipidemic individuals. Furthermore, our results suggest that reducing inflammation provides a potential mechanism by which the combination of n-3 PUFA and plant sterols are cardioprotective. (c) 2008 Elsevier Ireland Ltd. All rights reserved.

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 Adiponectin and leptin in ischemic stroke
 Adiponektyna i leptyna a udar niedokrwieny mozgu

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Abdominal obesity becomes very significant health's problem, especially because it is connected with pathogenesis of cardiovascular diseases. Adipose tissue is not only a store of excess energy but a hormonally active system too. The substances produced by adipose tissue are adipocytokines. Two of them are leptin and adiponectin. Adiponectin levels are inversely related to the adiposity degree, despite of adipose tissue is only source of it. Concentrations of adiponectin have been reported to be decreased in patients with coronary artery diseases, type II diabetes mellitus, hypertension and dyslipidemia patients in some insulin resistant states. It takes part in processes regulate glucose and lipid metabolism and it has anti-inflammatory and antiatherogenic properties. Adiponectin has a potential protective ability towards to cardiovascular diseases. Positive correlation with degree of adiposity has been reported for leptin - hormone involved in the regulation of food intake and energy expenditure. Leptin exerts many potentially atherogenic effects. It has been reported to influence on arterial hypertension, endothelial dysfunction, platelet aggregation, insulin resistant and activation of sympathetic system. In this way it can play very important role in development of stroke. Recent studies suggest that adiponectin and leptin may play an important role in obesity-associated cerebrovascular diseases. There is still too little evidence to say that these two hormones are independent marks of ischemic stroke and confirm their role in stroke pathogenesis. (c) Aktualn Neurol

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Adiponectin-adipocytes-derived hormone and its multidirectional function

Adiponektyna-hormon adipocytarny i jego wieloukladowe znaczenie
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LANGUAGE: Polish SUMMARY LANGUAGE: Polish; English

NUMBER OF REFERENCES: 65

Increasing morbidity and mortality from cardiovascular and neoplastic disorders is the effect of more frequent problem of obesity; insulinresistance and type 2 diabetes; not only in developed countries but in developing as well. Excess adipose tissue in particular compartments of human body is connected with numerous risk factors of atherosclerosis such as hyperinsulinaemia, hypertension, dyslipidaemia, dysregulations of glucose metabolism. Several studies that have been conducted so far, have focused on the pathomechanism of mentioned disorders with a particular attention paid to metabolism of adipocytes. Plasma proteins derived from adipocytes have been observed, especially the one called adiponectin. Adiponectin is the protein product of apM1 gene expression. Identified receptors of adiponectin: AdipoR1 and AdipoR2 are localised not only on adipocytes but also on hepatocytes and skeletal myocytes. Activation of the pleiotropic enzyme AMP-dependent protein kinase

is integral to the signalling intracellular effects of adiponectin. The potential antiinflammatory and antiatherosclerotic effects of adiponectin activity, as well as inhibition of neoplastic transformation originate from its influence on cellular adhesion molecules presentation and cytokines and growth factors production. Adiponectin, adipocytes-derived protein, appears in circulation in a form of hexamer or multimer. Considering the current data, adiponectin is an integral part of several signalling pathways of alive cell. There is obvious necessity of further researches on the precise role of adiponectin and prospective use of gained knowledge in clinical practice.

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0082546606 EMBASE No: 2008378719
Current studies on therapeutic approaches for ischemia/reperfusion injury in steatotic livers

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NUMBER OF REFERENCES: 86

Steatotic livers are particularly vulnerable to ischemia/reperfusion (I/R) injury, resulting in poor outcomes following liver surgery and transplantation. Therapeutic approaches for I/R injury in steatotic livers are currently under intensive investigation. This review summarizes and discusses the approaches developed during the last few years to prevent hepatic I/R injury in steatotic livers. Among the proposed approaches,

ischemic preconditioning and intermittent clamping are the two most promising approaches that have been applied in some clinical centers for liver surgery and transplantation, but most of others have not reached clinical application yet. (c) 2008 The Japan Society of Hepatology.

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0082522990 EMBASE No: 2008316157
Rimonabant in rats with a metabolic syndrome: Good news after the depression
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NUMBER OF REFERENCES: 12

The synthetic cannabinoid CB SUB 1 receptor antagonist rimonabant (sold in the United Kingdom under the brand name Acomplia) was reported to improve the profile of cardiovascular risk factors in obese patients with the metabolic syndrome, a cluster of metabolic disorders that often precedes the onset of type II diabetes. Rimonabant is shown in the current issue of British Journal of Pharmacology to attenuate weight gain in Zucker rats, an experimental model of insulin resistance. Neutrophil and monocyte counts were lowered by rimonabant administration. Both platelet activation (by ADP) and aggregation (in response to thrombin) were inhibited. Circulating pro-inflammatory cytokine levels (monocyte chemotactic protein 1, MCP1 and Regulated upon Activation, Normal T-cell Expressed and

Secreted, RANTES) were also reduced. Furthermore, fibrinogen levels returned to normal. These favourable anti-inflammatory and anti-thrombotic actions imply for rimonabant a peripheral, direct action on some cardiovascular risk factors. (c) 2008 Nature Publishing Group All rights reserved.

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0082485431 EMBASE No: 2008329961
Genetic epidemiology of diabetic retinopathy
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URL: <http://www.expert-reviews.com/doi/pdf/10.1586/17469899.3.2.165>
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 128

Diabetic retinopathy (DR) is the leading cause of vision loss and blindness among adults in developed countries. Despite intensive antidiabetic treatment, the global prevalence of DR is growing due to an increasing incidence and prolonged survival of diabetic individuals. As a complex disease, DR is a consequence of multiple interactions between environmental and genetic factors. Racial differences in incidence, familial aggregation and candidate gene association studies suggest that genetic factors play a role in the etiology of DR. Despite approximately 30 years of research, the molecular genetics of DR is still in its infancy owing to the low quality of most research studies. To date, only a few genetic markers from one gene, AKR1B1 (aldehyde reductase) - the

first enzyme involved in the polyol pathway of glucose metabolism that converts glucose into sorbitol - have been identified. Successful genetic epidemiology requires a high sample size, longitudinal designs, analysis of large haplotype blocks integrated by tag single nucleotide polymorphisms identified by means of human haplotype map (HapMap) data, genome-wide association studies using high density microarray-based single nucleotide polymorphism genotyping and pharmacogenomic approaches. An urgent move from old genetics to modern genomics is necessary to boost the ability to identify genes contributing to the development and progression of DR.
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0082269058 EMBASE No: 2008063117
Effects of olmesartan on endothelial function
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It is well established that a functional endothelium contributes to maintain cardiovascular homeostasis mainly through the activity of endothelium-derived nitric oxide (NO). However, in the presence of proatherogenic risk factors including hypertension, diabetes mellitus and hypercholesterolaemia, the bioavailability of NO is reduced. This condition

is defined as endothelial dysfunction and characterised by vasoconstriction, platelet aggregation, leucocyte adhesion and smooth muscle cell proliferation. A reduced availability of NO is mainly due to an increase in reactive oxygen species (ROS) production, which is responsible for NO breakdown. A large body of evidence indicates that, especially under pathological conditions, the activity of the renin-angiotensin system (RAS) is associated with angiotensin II (Ang II)-mediated ROS production, thus unbalancing endothelial function and leading to progressive vascular disease. The action of RAS is mostly linked to the downstream effects of the binding with the Ang II subtype 1 receptors (AT1). Therefore, selective RAS blockade with angiotensin receptor blockers (ARBs) is able to restore endothelial function in patients with cardiovascular risk factors. Olmesartan, an effective ARB, beyond its blood-pressure lowering effect, has been reported to affect the redox state of the vessel wall by restoring NO availability under different pathological conditions. Furthermore, it has been described that olmesartan exerts anti-inflammatory effects and increases endothelial progenitor cells. This article reviews the evidence linking olmesartan to vascular endothelial protection and examines the possibility that this effect translates to beneficial clinical properties of this ARB. (c) 2007 Adis Data Information BV. All rights reserved.

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0081835597 EMBASE No: 2007269702

Primary culture of human omental preadipocytes and study of their biological properties

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NUMBER OF REFERENCES: 12

Objective: To develop a primary culture method of human omental preadipocytes and to study their biological properties, such as hyperplasia, hypertrophy and endocrine secretion of human visceral adipose tissue. Methods: Using enzyme-digesting method, fibroblast-like cells from the human omental adipose tissues were cultured. The morphological changes of the cultured cells were observed and the growth curve was drawn by MTT method. The intracytoplasmic lipid of the cultured cells was determined by oil red O staining. The leptin and adiponectin levels in the culture supernatants were measured by ELISA. Results: The cultured fibroblast-like cells were homogeneous. Proliferation of cells began at the 3 rd day and the cell numbers increased in indicial way from the 3 rd day to the 9 th day. The doubling time of cells was about 60 hours. During the process of induction by conditional medium, the cells became round and larger, and more adipose droplets were aggregated. On the 21 st day, more than 90% of the cells became adipocytes. Leptin secretion was detected at low level in the preadipocytes and continuously increased during differentiation, with a peak on day 17. It remained constant from day 17 onward. Unlike leptin, adiponectin secretion was not detected until day 7 after induction, when differentiated adipocytes had already been observed. Its secretion increased dramatically between days 7 and 17, and reached a maximum level on day 17, but had a significant reduction on day 21. Extraction of intracytoplasmic lipid stained with oil red O and detection of leptin and adiponectin both verified the isolated cells were preadipocytes functioning actively. Conclusion: A human omental preadipocytes model has been established and different secretion patterns of leptin and adiponectin secretion related to preadipocyte differentiation has been characterized. Adiponectin may be proposed as a specific marker for preadipocyte differentiation.

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Targeting adiponectin for cardioprotection
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Adiponectin is an adipose tissue-derived plasma protein which has a
reduced concentration in subjects with obesity-related diseases.
Adiponectin has anti-diabetic and anti-inflammatory characteristics,
which lead to beneficial actions on various obesity-linked
complications.
Recent experimental findings have shown that adiponectin contributes
to protection against cardiac remodelling after pressure overload and
cardiac injury following ischaemia-reperfusion. Thus, adiponectin
could emerge as a potential cardioprotective agent for the treatment
of
several pathological heart conditions. (c) 2006 Informa UK Ltd.

3/7/87 (Item 9 from file: 72)
DIALOG(R)File 72:EMBASE
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0081172346 EMBASE No: 2006234601
Adiponectin: Vascular protection from the fat?
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Arteriosclerosis, Thrombosis, and Vascular Biology (Arterioscler. Thromb. Vasc. Biol.) (United States) February 1, 2006, 26/2 (235-236)

CODEN: ATVBF ISSN: 1079-5642

PUBLISHER ITEM IDENTIFIER: 0004360520060200000001

DOI: 10.1161/01.ATV.0000200222.55680.df

DOCUMENT TYPE: Journal; Editorial RECORD TYPE: Citation

LANGUAGE: English

NUMBER OF REFERENCES: 19

3/7/88 (Item 10 from file: 72)
DIALOG(R)File 72:EMBASE
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0081136820 EMBASE No: 2006198770

Adipocyte: A potential target for the treatment of atherosclerosis
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Medical Hypotheses (Med. Hypotheses) (United Kingdom) June 16, 2006, 67/1 (82-86)

CODEN: MEHYD ISSN: 0306-9877

PUBLISHER ITEM IDENTIFIER: S0306987706000508

DOI: 10.1016/j.mehy.2005.12.042

DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract

LANGUAGE: English SUMMARY LANGUAGE: English

NUMBER OF REFERENCES: 48

Obesity is an independent risk factor for coronary heart disease, whereas the underlying mechanisms have not been fully elucidated. Adipocytes may produce various adipokines with favorable and unfavorable cardiovascular effects. The dysregulated secretion of adipokines by adipocytes may contribute to obese associated atherosclerosis. Adipocytes can also function as phagocytes to uptake and degrade oxidized low-density lipoprotein (Ox-LDL), suggesting that adipocytes possibly involve in

clearance of Ox-LDL in blood. The dysfunctional adipocytes might be implicated in the atherogenesis. Some cardioprotective drugs mediate their cardiovascular benefits partly through their direct beneficial effects on adipocytes. Therefore, we hypothesized that adipocytes might be potential target for the treatment of atherosclerosis. (c) 2006 Elsevier Ltd. All rights reserved.

3/7/89 (Item 11 from file: 72)
DIALOG(R)File 72:EMBASE
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0081048235 EMBASE No: 2006108250
Linking inflammation and atherogenesis: Soluble markers identified for the detection of risk factors and for early risk assessment
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Clinica Chimica Acta (Clin. Chim. Acta) (Netherlands) April 1, 2006,
366/1-2 (74-80)
CODEN: CCATA ISSN: 0009-8981
PUBLISHER ITEM IDENTIFIER: S0009898105006352
DOI: 10.1016/j.cca.2005.10.016
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 56

Increasing evidence has shown that atherogenesis is not only caused by hypercholesterolemia. Several risk factors including abdominal obesity, dyslipidemia, hyperglycemia, bacterial and viral infection, hyperhomocysteinemia have been identified recently, all mediated through

inflammation, which can lead to atherosclerosis. Several events have also been identified to be involved in the overall inflammation reaction in the blood vessel which include endothelium dysfunction, expression of adhesion molecules, recruitment of leukocytes to the injured endothelium, migration of monocytes to the arterial intima, and transformation of monocytes to macrophages. In order to facilitate the assessment of early risk for atherogenesis we have made an effort in this review to identify soluble markers that will allow the detection of these risk factors and the identification of associated inflammation events. Since early risks for atherogenesis are largely preventable with dietary modification and lifestyle changes, capable of detecting early risks by monitoring soluble risk markers is conceivably important for asymptomatic individuals to avoid serious or fatal consequences of atherosclerosis. These soluble markers should also be useful for monitoring the effectiveness of intervention and for the identification of therapeutic targets. (c) 2005 Elsevier B.V. All rights reserved.

3/7/90 (Item 12 from file: 72)
DIALOG(R)File 72:EMBASE
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0081048230 EMBASE No: 2006108245
Genomic variants in polycystic ovary syndrome
Luque-Ramirez M.; San Millan J.L.; Escobar-Morreale H.F.
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Clinica Chimica Acta (Clin. Chim. Acta) (Netherlands) April 1, 2006,
366/1-2 (14-26)
CODEN: CCATA ISSN: 0009-8981
PUBLISHER ITEM IDENTIFIER: S0009898105006364
DOI: 10.1016/j.cca.2005.10.017

DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract

LANGUAGE: English SUMMARY LANGUAGE: English

NUMBER OF REFERENCES: 181

The polycystic ovary syndrome (PCOS) is a common disorder in premenopausal women, characterized by the presence, among other traits, of hyperandrogenism, insulin resistance, and hyperinsulinism. The familial aggregation of PCOS lead the interest to the molecular genetic basis of this syndrome, especially to the genes encoding proteins involved in androgen synthesis and the regulation of insulin synthesis and action. Considering the relationship between insulin resistance and chronic inflammation, and the clustering of inflammatory markers in PCOS patients, recent studies focused on the involvement of proinflammatory genotypes on the pathogenesis of PCOS. Mounting evidence suggest at present a complex model of inheritance for PCOS, in which predisposing and protecting genomic variants interact with environmental factors such as obesity and a sedentary lifestyle, finally leading to the classic phenotype of this syndrome. Moreover, the association of hyperandrogenism, insulin resistance and chronic inflammation raised the possibility of an increase risk of cardiovascular disease in women suffering from PCOS. In the present review we will summarize the most important findings published to date regarding the molecular genetic mechanisms underlying the association of PCOS with insulin resistance and chronic inflammation, and the possible interaction of these mechanisms with environmental factors. (c) 2005 Elsevier B.V. All rights reserved.

3/7/91 (Item 13 from file: 72)

DIALOG(R)File 72:EMBASE

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0080482469 EMBASE No: 2005126627

Cardiovascular and metabolic disease: New opportunities for therapy
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Current Opinion in Pharmacology (Curr. Opin. Pharmacol.) (United Kingdom) April 1, 2005, 5/2 SPEC. ISS. (119-121)

CODEN: COPUB ISSN: 1471-4892

PUBLISHER ITEM IDENTIFIER: S1471489205000184

DOI: 10.1016/j.coph.2005.02.002

DOCUMENT TYPE: Journal; Editorial RECORD TYPE: Citation

LANGUAGE: English

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DIALOG(R)File 72:EMBASE

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0080189180 EMBASE No: 2004368723

Tumor necrosis factor and its potential role in insulin resistance and

nonalcoholic fatty liver disease

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Clinics in Liver Disease (Clin. Liver Dis.) (United States)

August 1,

2004, 8/3 (619-638)

CODEN: CLDIF ISSN: 1089-3261

PUBLISHER ITEM IDENTIFIER: S108932610400042X

DOI: 10.1016/j.cld.2004.04.012

DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract

LANGUAGE: English SUMMARY LANGUAGE: English

NUMBER OF REFERENCES: 120

Adipose tissue is an important source of TNF and molecules that regulate

TNF activity. Visceral adiposity, in particular, promotes a profile of adipokine expression (ie, low adiponectin, high TNF) that permits high TNF activity and insulin resistance. At the cellular level, TNF-dependent activation of stress-related kinases inhibits insulin signal

transduction, causing cellular insulin resistance. Some of the same kinases

also promote further production of TNF, perpetuating a self-re-enforcing, positive feedback mechanism for sustained TNF activity and chronic insulin resistance. Increased TNF inhibits the hepatic actions of adiponectin, increasing hepatic fatty acid uptake while reducing hepatic fatty acid oxidation and triglyceride export. In aggregate, these responses cause the net accumulation of fat within hepatocytes (ie, hepatic steatosis). In mice, the increased delivery of fatty acids to hepatocytes is sufficient to induce hepatic insulin resistance. Consistent with this finding, it has been shown that TNF-dependent inhibition of adiponectin blocks normal insulin-mediated suppression of hepatic glucose output, causing hyperglycemia. This hyperglycemia, in turn, stimulates hyperinsulinemia. Hyperinsulinemia also attenuates the further propagation of insulin-initiated signals (ie, hyperinsulinemic insulin resistance). Given this evidence that fatty livers participate in the pathogenesis of type 2 diabetes, therapy to prevent or reverse NAFL seems appropriate. Increased TNF also promotes hepatocyte death by inducing molecules that cause apoptosis and molecules that cause necrosis. Which death response occurs in any given hepatocyte may be determined by the cellular ATP level (low ATP favors necrosis over apoptosis). In addition, the increased TNF activity induces the production of other cytokines and chemokines that promote the hepatic accumulation of inflammatory cells. NASH is the histologic manifestation of increased rates of hepatocyte death and associated inflammatory cell infiltration. Studies in experimental animals prove that TNF-related insulin resistance causes NASH and alcohol-induced steatohepatitis (ASH), because treatment with agents that directly inhibit TNF activity improve insulin resistance, NASH, and ASH. Similarly, treatment with insulin sensitizers reduces TNF activity and improves NASH and ASH. Recent evidence raises the ominous possibility that NASH may be a premalignant condition. Chronic exposure to TNF induces hepatic oxidant stress, and the latter reduces the proliferative activity of mature hepatocytes. The increased death rates and reduced proliferative activity of mature hepatocytes, in turn, promote liver progenitor populations to expand. This may set the stage for subsequent hepatic neoplasia, because in people and experimental animals, the incidence of

hepatocellular carcinoma is increased in settings that promote the accumulation of hepatic progenitor cells. Nonalcoholic steatohepatitis also appears to play a permissive role for hepatic fibrosis, because the incidence of cirrhosis is increased in individuals with NASH compared with those with simple NAFL. Studies in people and experimental animals, however, also clearly demonstrate that NASH is not sufficient to assure the development of cirrhosis. Indeed many (and perhaps most) individuals with NASH never develop cirrhosis. Efforts to explain interindividual variability in hepatic fibrosis have identified factors that act on stellate cells to regulate hepatic fibrogenesis. These include leptin, certain neurotransmitters (eg, norepinephrine, angiotensin, and acetylcholine) and other cytokines (eg, IL-10 and TGF-beta). Interactions between TNF and each of these factors have been demonstrated, as have interactions among the factors themselves. Additional research is needed to clarify the exact milieu required to drive a sustained fibrogenic response during liver injury. This knowledge has important clinical implications, because it will help identify the subset of NASH patients who are destined to become cirrhotic and therefore, most worthy of aggressive clinical intervention.

3/7/93 (Item 15 from file: 72)
DIALOG(R)File 72:EMBASE
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0080056461 EMBASE No: 2004241636

The chronic inflammatory hypothesis for the morbidity associated with morbid obesity: Implications and effect of weight loss
Cottam D.R.; Mattar S.G.; Barinas-Mitchell E.; Eid G.; Kuller L.; Kelley D.E.; Schauer P.R.

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Obesity Surgery (Obes. Surg.) (United States) May 1, 2004, 14/5
(589-600)

CODEN: OBSUE ISSN: 0960-8923

DOI: 10.1381/096089204323093345

DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract

LANGUAGE: English SUMMARY LANGUAGE: English

NUMBER OF REFERENCES: 185

Background: Obesity is a worldwide pandemic that causes a multitude of co-morbid conditions. However, there has been slow progress in understanding the basic pathophysiology that underlies co-morbid conditions associated with obesity. Recently, there has been intense interest in the role of inflammation in obesity. Using the inflammatory hypothesis, many of the mechanisms by which co-morbid conditions are associated with obesity are being elucidated. Methods: We searched the literature and reviewed all relevant articles. We focused on hormones and cytokines that have been associated with other inflammatory conditions such as sepsis and systemic inflammatory response syndrome. Findings: Angiotensinogen (AGT), transforming growth factor beta (TGFbeta), tumor necrosis factor alpha (TNFalpha), and interleukin six (IL-6) are all elevated in obesity and correlate with several markers of adipocyte mass. These mediators have detrimental effects on hypertension, diabetes, dyslipidemia, thromboembolic phenomena, infections, and cancer. Weight loss results in a reduction of inflammatory mediators and a diminution of the associated co-morbid conditions. Conclusions: The success of weight loss surgery in treating the complications associated with obesity is most probably related to the reduction of inflammatory mediators. While some aspects of bariatric physiology remain unclear, there appears to be a strong association between obesity and inflammation, thereby rendering obesity a chronic inflammatory state. A clearer understanding of the physiology of obesity will allow physicians who treat the obese to develop better strategies to promote weight loss and improve the well-being of millions of individuals.

3/7/94 (Item 16 from file: 72)

DIALOG(R)File 72:EMBASE

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0079995567 EMBASE No: 2004180717

Techniques: Cardiovascular pharmacology and drug discovery in the
21st
century

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Trends in Pharmacological Sciences (Trends Pharmacol. Sci.)
(United

Kingdom) April 1, 2004, 25/4 (225-233)

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PUBLISHER ITEM IDENTIFIER: S0165614704000641

DOI: 10.1016/j.tips.2004.02.013

DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract

LANGUAGE: English SUMMARY LANGUAGE: English

NUMBER OF REFERENCES: 88

The latter half of the 20th century has been characterized by
pharmacologists as the 'age of the receptor', an era in which the
bioassay,
that stalwart of classical pharmacology, has played a seminal role in
identifying novel cardiovascular medicines. In this article, we ask
what,
if anything, has changed in the pharmacologist's approach to
discovering
novel cardiovascular drugs on this, the 25th anniversary of the
inaugural
publication of Trends in Pharmacological Sciences.

3/7/95 (Item 1 from file: 144)

DIALOG(R)File 144:Pascal

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19079071 PASCAL No.: 09-0160178

Comparison of Immunoassays for the Selective Measurement of Human
High-Molecular Weight Adiponectin

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Journal: Clinical chemistry : (Baltimore, Md.), 2009, 55 (3)
568-572

ISSN: 0009-9147 CODEN: CLCHAU Availability: INIST-7603;
354000187401760290

No. of Refs.: 17 ref.

Document Type: P (Serial) ; A (Analytic)

Country of Publication: United States

Language: English

BACKGROUND: Adiponectin is an adipocyte-derived hormone circulating in different multimer complexes. The high-molecular-weight (HMW) complex is likely the active form of this protein and has been recognized

as a risk marker for type 2 diabetes and coronary artery disease (CAD).

Because quantification of HMW adiponectin by Western blot analysis is time-consuming, novel ELISAs have been developed to simplify measurements

in clinical research. However, these enzyme immunoassays have not been

cross-validated in larger patient groups. We evaluated 2 individual ELISA

systems by comparison to Western blotting for measurement of the

distribution of HMW adiponectin in healthy individuals and patients with CAD and type 2 diabetes. METHODS: We measured HMW adiponectin in 204 individuals (83 CAD patients, 81 type 2 diabetes patients, and 40

healthy controls). Correlations, range of agreement, and imprecision of HMW

concentrations obtained using 2 commercial ELISAs (#1, ALPCO Diagnostics;

#2, Millipore) were evaluated by comparison with quantitative Western

blotting. RESULT: Adiponectin results of the ELISAs were significantly correlated with those obtained by Western blotting (both $r >$

0.75, $P < 0.001$). Deming regression and Bland-Altman analyses indicated

high agreement among the 3 immunoassays. The median difference between HMW

adiponectin concentrations measured by ELISA and by Western blot was +0.4 mg/L for ELISA #1 and -0.4 mg/L for ELISA #2 with 95% of value

differences < 3 mg/L. CONCLUSIONS: Selective measurement of HMW

adiponectin by ELISA is feasible; however, individual differences among immunoassays must be considered. The evaluated ELISAs exhibit

analytical characteristics that allow their use as equivalent for Western

blot analysis in larger clinical and epidemiological groups.

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3/7/96 (Item 1 from file: 154)
DIALOG(R)File 154:MEDLINE(R)
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Available on
03/01/09

Reduced albuminuria with sarpogrelate is accompanied by a decrease in monocyte chemoattractant protein-1 levels in type 2 diabetes.

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States) Mar 2008, 3 (2) p362-8, ISSN 1555-905X--Electronic Journal Code: 101271570

Publishing Model Print-Electronic

Document type: Journal Article; Randomized Controlled Trial

Languages: ENGLISH

Main Citation Owner: NLM

Other Citation Owner: NLM

Record type: MEDLINE; Completed

BACKGROUND AND OBJECTIVES: Sarpogrelate has been shown to reduce albuminuria in diabetic nephropathy. For examination of whether this is

based on the same mechanisms as angiotensin II receptor blockers or

thiazolidinedione, effects of sarpogrelate on atherosclerotic inflammatory

molecules and their relations to albuminuria in patients who had diabetes

and had already been treated with angiotensin II receptor blockers and with

or without thiazolidinedione were examined. DESIGN, SETTING, PARTICIPANTS,

& MEASUREMENTS: Forty patients who had diabetes with nephropathy and

arteriosclerosis obliterans and had already been treated with angiotensin

II receptor blocker (n = 40) were randomly assigned to sarpogrelate (300

mg/d; n = 20) or aspirin group (100 mg/d; n = 20). Plasma monocyte

chemoattractant protein-1 and urinary albumin-to-creatinine ratio and monocyte chemoattractant protein-1 were measured at baseline and 16 wk after administration. RESULTS: Only the sarpogrelate group showed increases in plasma adiponectin and decreases in both plasma and urinary monocyte chemoattractant protein-1 and albumin-to-creatinine ratio levels. Moreover, percentage change of monocyte chemoattractant protein-1 level correlated positively to that of albumin-to-creatinine ratio. Even when the sarpogrelate group was further divided into two groups with (n = 9) or without thiazolidinedione (n = 11), changes in monocyte chemoattractant protein-1 or albumin-to-creatinine ratio did not differ. CONCLUSIONS: Sarpogrelate can reduce albuminuria and plasma and urinary monocyte chemoattractant protein-1 levels while increasing plasma adiponectin in diabetic nephropathy. These effects seem to be mediated via mechanisms that are different from those of angiotensin II receptor blocker or thiazolidinedione.

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3/7/97 (Item 2 from file: 154)
DIALOG(R)File 154:MEDLINE(R)
(c) format only 2009 Dialog. All rts. reserv.

17390366 PMID: 16936907

Adiponectin added into the plasma of healthy probands does not affect platelet aggregability.

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Biomedical papers of the Medical Faculty of the University Palacky,

Olomouc, Czechoslovakia (Czech Republic) Jul 2006, 150 (1) p89-90,

ISSN 1213-8118--Print Journal Code: 101140142

Publishing Model Print

Document type: In Vitro; Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Six healthy non-obese probands without medical therapy and history of disease were tested. In all of them platelet aggregability with addition of human recombinant adiponectin in different concentrations (100; 75; 50 and 25 ng/l) were measured. It is concluded that increased level of adiponectin has no significant antiaggregation effect on platelets from individuals without hypoadiponectinemia.

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Record Date Completed: 20070823

3/7/98 (Item 3 from file: 154)

DIALOG(R)File 154:MEDLINE(R)

(c) format only 2009 Dialog. All rts. reserv.

16729453 PMID: 16123486

Osteoprotegerin is associated with silent coronary artery disease in high-risk but asymptomatic type 2 diabetic patients.

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Cristol Jean Paul; Dupuy Anne Marie

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Diabetes care (United States) Sep 2005, 28 (9) p2176-80, ISSN

0149-5992--Print Journal Code: 7805975

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

OBJECTIVE: Osteoprotegerin (OPG) is an inhibitor of osteoclastogenesis, which has been recently involved in atherosclerosis. The relationship between coronary atherosclerosis and OPG has never been studied in asymptomatic type 2 diabetic patients. RESEARCH DESIGN AND METHODS: This is a nested case-control study; 162 asymptomatic type 2 diabetic patients were evaluated for silent myocardial ischemia using stress myocardial perfusion imaging; of 50 patients with positive results, 37 underwent coronary angiography, 20 of whom showed significant coronary artery disease (CAD

group). Of 112 patients without silent myocardial ischemia, 20 subjects (NO-CAD group) were selected and matched by age and sex to patients with CAD. OPG, C-reactive protein, adiponectin, lipoprotein(a), albuminuria, and classical risk factors were measured. RESULTS: The percentages of subjects with OPG levels above median and with nephropathy were higher in the CAD group than in the NO-CAD group (70 vs. 25%, $P = 0.004$ and 50 vs. 5%, $P = 0.001$, respectively). LDL cholesterol levels were higher and HDL cholesterol levels lower in the CAD compared with the NO-CAD group ($P = 0.033$ and $P = 0.005$, respectively). No other variables were associated with CAD. Logistic regression analysis showed that OPG values above median (odds ratio 8.31 [95% CI 1.18-58.68], $P = 0.034$) and nephropathy (21.98 [1.24-388.36], $P = 0.035$) were significant independent predictors of asymptomatic CAD in type 2 diabetic patients. CONCLUSIONS: Our investigation reports the first evidence of an independent association of OPG with asymptomatic CAD in type 2 diabetic patients. The results of this nested case-control study with 20 cases need to be confirmed in a larger population.
Record Date Created: 20050826
Record Date Completed: 20051107

3/7/99 (Item 4 from file: 154)
DIALOG(R)File 154:MEDLINE(R)
(c) format only 2009 Dialog. All rts. reserv.

16277342 PMID: 15599338
[Adiponectin gene polymorphism and protein dysfunction in the development of insulin resistance]
Polimorfizm genu i zaburzenia funkcjonalne adiponektyny jako jedna z przyczyn rozwoju opornosci na insuline.
Karbowska Joanna; Warczak Elzbieta; Kochan Zdzislaw
Katedra Biochemii Akademii Medycznej w Gdansk.
Post py higieny i medycyny doswiadczalnej (Online) (Poland)
2004, 58
p449-57, ISSN 1732-2693--Electronic Journal Code: 101206517
Publishing Model Print

Document type: English Abstract; Journal Article; Review

Languages: POLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Adiponectin, an adipocyte-secreted protein encoded by the ACDC gene (also known as APM1), has been shown to play an important role in the regulation of fatty acid and glucose metabolism in liver and muscle, where it modulates insulin sensitivity. Adiponectin enhances fatty acid oxidation in liver and muscle, thus reducing triglyceride content in these tissues. Moreover, it stimulates glucose utilization in muscle and inhibits glucose production by the liver, consequently decreasing blood glucose levels. Plasma adiponectin levels are positively correlated with insulin sensitivity in humans. Circulating adiponectin forms a wide range of multimers. Mutations in the ACDC gene result in an impaired multimerization and/or impaired secretion of adiponectin from adipocytes, both linked to the development of insulin resistance and type II diabetes. This review focuses on the molecular mechanisms underlying hypoadiponectinemia associated with the diabetic phenotype. We further discuss the more recent findings that implicate adiponectin multimer formation as an important feature of the biological function of this adipocyte-derived hormone. (54 Refs.)

Record Date Created: 20041215

Record Date Completed: 20060421

3/7/100 (Item 1 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

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150463748 CA: 150(22)463748k JOURNAL

The effects of pitavastatin, eicosapentaenoic acid and combined therapy

on platelet-derived microparticles and adiponectin in hyperlipidemic, diabetic patients

AUTHOR(S): Nomura, Shosaku; Inami, Norihito; Shouzu, Akira; Omoto, Seitarou; Kimura, Yutaka; Takahashi, Nobuyuki; Tanaka, Atsushi; Urase, Fumiaki; Maeda, Yasuhiro; Ohtani, Hajime; Iwasaka, Toshiji

LOCATION: Division of Hematology, Kishiwada City Hospital, Kishiwada, Japan,

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PUBLISHER: Informa Healthcare

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derived

microparticle hyperlipidemia diabetes antiatherosclerotic

DESCRIPTORS:

Antiarteriosclerotics...

antiatherosclerotics; pitavastatin, eicosapentaenoic acid and
combined

therapy on platelet-derived microparticles and adiponectin

High-density lipoproteins...

HDL cholesterol, HDL-C; pitavastatin, eicosapentaenoic acid and
combined therapy on platelet-derived microparticles and

adiponectin

Low-density lipoproteins...

LDL cholesterol, LDL-C; pitavastatin, eicosapentaenoic acid and
combined therapy on platelet-derived microparticles and

adiponectin

Diabetes mellitus...

non-insulin-dependent; pitavastatin, eicosapentaenoic acid and
combined

therapy on platelet-derived microparticles and adiponectin

Adiponectins... Atherosclerosis... CD40(antigen)... Combination

chemotherapy... Diabetic angiopathy... Human... Hyperlipidemia...

Platelet

aggregation inhibitors... Triglycerides...

pitavastatin, eicosapentaenoic acid and combined therapy on
platelet-derived microparticles and adiponectin

CAS REGISTRY NUMBERS:

57-88-5 biological studies, LDL and HDL cholesterol; pitavastatin,
eicosapentaenoic acid and combined therapy on platelet-derived
microparticles and adiponectin

10417-94-4 147511-69-1 pitavastatin, eicosapentaenoic acid and
combined

therapy on platelet-derived microparticles and adiponectin

3/7/101 (Item 2 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

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148423018 CA: 148(19)423018z JOURNAL

Relationship of multimers and genetic polymorphism of adiponectin
with

type 2 diabetes

AUTHOR(S): Sun, Hong; Tan, Yuanming; Liu, Zhaoqian

LOCATION: Institute of Clinical Pharmacology, Central South
University,

Changsha, Peop. Rep. China, 410078

JOURNAL: Guoji Bingli Kexue Yu Linchuang Zazhi (Gujia Bingli Kexue
Yu

Linchuang Zazhi) DATE: 2006 VOLUME: 26 NUMBER: 5 PAGES: 432-435
CODEN: GBKYAR ISSN: 1673-2588 LANGUAGE: Chinese PUBLISHER: Guoji
Bingli Kexue Yu Linchuang Zazhi Bianjibu

SECTION:

CA214000 Mammalian Pathological Biochemistry

IDENTIFIERS: review adiponectin single nucleotide polymorphism
multimer

type 2 diabetes

DESCRIPTORS:

Diabetes mellitus...

non-insulin-dependent; relationship of multimers and genetic
polymorphism of adiponectin with type 2 diabetes

Adiponectins... Single nucleotide polymorphism...

relationship of multimers and genetic polymorphism of adiponectin
with

type 2 diabetes

3/7/102 (Item 3 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

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146314021 CA: 146(16)314021h JOURNAL

Adiponectin inhibits osteoclast formation stimulated by
lipopolysaccharide from Actinobacillus actinomycetemcomitans

AUTHOR(S): Yamaguchi, Noboru; Kukita, Toshio; Li, Yin-Ji; Argueta,
Jose

Guillermo Martinez; Saito, Toshiyuki; Hanazawa, Shigemasa; Yamashita,
Yoshihisa

LOCATION: Department of Preventive Dentistry, Kyushu University
Faculty

of Dental Science, Fukuoka, Japan,

JOURNAL: FEMS Immunol. Med. Microbiol. (FEMS Immunology and Medical
Microbiology) DATE: 2007 VOLUME: 49 NUMBER: 1 PAGES: 28-34 CODEN:
FIMIEV ISSN: 0928-8244 LANGUAGE: English PUBLISHER: Blackwell
Publishing

Ltd.

SECTION:

CA214003 Mammalian Pathological Biochemistry

IDENTIFIERS: adiponectin osteoclast lipopolysaccharide
Actinobacillus

RANKL iNOS TLR4 NFkappaB, periodontal disease adiponectin
osteoclast

lipopolysaccharide Actinobacillus RANKL

DESCRIPTORS:

Adiponectins... Aggregatibacter actinomycetemcomitans...

Periodontium,disease... Osteoclast...

adiponectin inhibited osteoclast formation stimulated by

lipopolysaccharide from Actinobacillus actinomycetemcomitans via
RANKL,

iNOS, TLR4, and NF- κ B

Lipopolysaccharides...

bacterial; adiponectin inhibited osteoclast formation stimulated
by
lipopolysaccharide from Actinobacillus actinomycetemcomitans via
RANKL,
iNOS, TLR4, and NF- κ B
Gene, animal...
iNOS; adiponectin inhibited osteoclast formation stimulated by
lipopolysaccharide from Actinobacillus actinomycetemcomitans via
RANKL,
iNOS, TLR4, and NF- κ B
Transcription factors...
NF- κ B (nuclear factor of κ light chain gene enhancer in
B-cells); adiponectin inhibited osteoclast formation stimulated by
lipopolysaccharide from Actinobacillus actinomycetemcomitans via
Toll-like receptors...
TLR-4; adiponectin inhibited osteoclast formation stimulated by
lipopolysaccharide from Actinobacillus actinomycetemcomitans via
RANKL,
iNOS, TLR4, and NF- κ B
CAS REGISTRY NUMBERS:
501433-35-8 207621-35-0 adiponectin inhibited osteoclast formation
stimulated by lipopolysaccharide from Actinobacillus
actinomycetemcomitans via RANKL, iNOS, TLR4, and NF- κ B
10102-43-9 biological studies, adiponectin inhibited osteoclast
formation
stimulated by lipopolysaccharide from Actinobacillus
actinomycetemcomitans via RANKL, iNOS, TLR4, and NF- κ B

3/7/103 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2009 American Chemical Society. All rts. reserv.

146050200 CA: 146(3)50200u PATENT
Pharmaceutical compositions of adiponectin variants and methods of
storage
INVENTOR(AUTHOR): Zalevsky, Jonathan; Thi Nguyen, Duc-Hanh; Moore,
Gregory L.; Ezhevsky, Sergei A.; Desjarlais, John R.; Chirino, Arthur
J.;
Cash, Darian; Bernett, Matthew J.
LOCATION: USA
PATENT: U.S. Pat. Appl. Publ. ; US 20060276395 A1 DATE: 20061207
APPLICATION: US 2006421061 (20060530) *US 2005PV642476 (20050107)
*US
2005PV650411 (20050203) *US 2005PV698358 (20050711) *US 2005PV720768
(20050926) *US 2005PV733137 (20051102) *US 2006328901 (20060109) *US
2006PV777825 (20060301) *US 2006PV781509 (20060309) *US 2006PV790220
(20060407)
PAGES: 79pp., Cont.-in-part of U.S. Ser. No. 328,901. CODEN: USXXCO
LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: 514012000

IPCR/8 + Level Value Position Status Version Action Source Office:
A61K-0038/17 A I F B 20060101 20061207 H US

SECTION:

CA263005 Pharmaceuticals
CA201XXX Pharmacology
CA203XXX Biochemical Genetics

IDENTIFIERS: adiponectin variant pharmaceutical compn storage

DESCRIPTORS:

Human...

adiponectin variants; pharmaceutical compns. of adiponectin
variants

and methods of storage

Drug delivery systems...

carriers; pharmaceutical compns. of adiponectin variants and
methods of

storage

Stability...

improved; pharmaceutical compns. of adiponectin variants and
methods of

storage

Solubility...

increased; pharmaceutical compns. of adiponectin variants and
methods

of storage

Aggregation...

low, after storage; pharmaceutical compns. of adiponectin
variants and

methods of storage

Protein sequences...

of adiponectin variants; pharmaceutical compns. of adiponectin
variants

and methods of storage

Adiponectins... Storage... Molecular cloning... Protein engineering...

pharmaceutical compns. of adiponectin variants and methods of
storage

Metabolic disorders...

treatment; pharmaceutical compns. of adiponectin variants and
methods

of storage

Buffers...

10 mM PO4, 150 mM NaCl, storage in; pharmaceutical compns. of
adiponectin variants and methods of storage

CAS REGISTRY NUMBERS:

7647-14-5 biological studies, 10 mM PO4, 150 mM NaCl buffer, storage
in;

pharmaceutical compns. of adiponectin variants and methods of
storage

916465-32-2 916465-33-3 916465-34-4 916465-35-5 916465-36-6

916465-37-7 916465-38-8 unclaimed protein sequence;

pharmaceutical

compns. of adiponectin variants and methods of storage

916465-15-1DP variants, amino acid sequence; pharmaceutical compns.
of

adiponectin variants and methods of storage

3/7/104 (Item 5 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

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145468690 CA: 145(24)468690s JOURNAL

High molecular weight adiponectin

AUTHOR(S): Horikoshi, Momoko

LOCATION: Grad. Sch. of Medicine, The Univ. of Tokyo, Tokyo, Japan,

JOURNAL: Igaku no Ayumi (Igaku no Ayumi) DATE: 2006 VOLUME: 217

NUMBER: 1 PAGES: 156-162 CODEN: IGAYAY ISSN: 0039-2359 LANGUAGE:

Japanese PUBLISHER: Ishiyaku Shuppan

SECTION:

CA214000 Mammalian Pathological Biochemistry

CA215XXX Immunochemistry

IDENTIFIERS: review adiponectin multimer diabetes insulin resistance
metabolic syndrome

DESCRIPTORS:

Cytokines...

adiponectin; relationship of high mol. wt. adiponectin with
insulin

resistance, diabetes and metabolic syndrome

Metabolic disorders...

metabolic syndrome X; relationship of high mol. wt. adiponectin
with

insulin resistance, diabetes and metabolic syndrome

Self-association...

multimerization; relationship of high mol. wt. adiponectin with
insulin

resistance, diabetes and metabolic syndrome

Diabetes mellitus... Human...

relationship of high mol. wt. adiponectin with insulin resistance,
diabetes and metabolic syndrome

CAS REGISTRY NUMBERS:

9004-10-8 biological studies, resistance; relationship of high mol.
wt.

adiponectin with insulin resistance, diabetes and metabolic
syndrome

3/7/105 (Item 6 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

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145181041 CA: 145(10)181041v JOURNAL

Adiponectin - a key adipokine in the metabolic syndrome

AUTHOR(S): Whitehead, J. P.; Richards, A. A.; Hickman, I. J.;
Macdonald,
G. A.; Prins, J. B.

LOCATION: Centre for Diabetes and Endocrine Research, Princess
Alexandra

Hospital, University of Queensland, Brisbane, Australia

JOURNAL: Diabetes, Obes. Metab. (Diabetes, Obesity and Metabolism)

DATE: 2006 VOLUME: 8 NUMBER: 3 PAGES: 264-280 CODEN: DOMEF6

ISSN:

1462-8902 LANGUAGE: English PUBLISHER: Blackwell Publishing Ltd.

SECTION:

CA202000 Mammalian Hormones

IDENTIFIERS: review adiponectin insulin vascular inflammation
metabolic

syndrome

DESCRIPTORS:

Heart...

adiponectin secretion and circulating levels reduced in patient
with
coronary artery disease

Diabetes mellitus... Pancreas...

adiponectin secretion and circulating levels reduced in patient
with
diabetes

Human...

adiponectin secretion and circulating levels reduced in patient
with
diabetes and coronary artery disease

Cytokines...

adiponectin; thiazolidinedione suppress insulin resistance and
inflammation via adiponectin regulation in human

Peroxisome proliferator-activated receptors...

α ; adiponectin receptor AdipoR2 was highly expressed in liver,
enhanced insulin sensitivity, reduced steatosis via activation of
AMPK

and increased peroxisome-proliferator-activated receptor- α

Artery,disease...

coronary; adiponectin secretion and circulating levels reduced in
patient with coronary artery disease

Metabolic disorders...

metabolic syndrome X; adiponectin improved hepatic insulin
sensitivity,

increased fuel oxidn., decreased vascular inflammation, suggest
adiponectin replacement therapy can be used to treat metabolic s

Disease,animal...

steatosis; adiponectin receptor, AdipoR2 highly expressed in
liver,

enhanced insulin sensitivity, reduced steatosis via activation of
AMPK

and increased peroxisome-proliferator-activated receptor- α

Blood vessel,disease... Inflammation...

vasculitis; adiponectin decreased vascular inflammation in human

Cadherins...

13; T-cadherin expressed in endothelium, smooth muscle and
identified

as adiponectin-binding protein with preference for HMW adiponectin
multimer in human

CAS REGISTRY NUMBERS:

9004-10-8 biological studies, adiponectin improved hepatic insulin sensitivity in diabetic patient

3/7/106 (Item 7 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

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144163988 CA: 144(10)163988j JOURNAL

Improvements in insulin resistance with weight loss, in contrast to rosiglitazone, are not associated with changes in plasma adiponectin or

adiponectin multimeric complexes

AUTHOR(S): Abbasi, Fahim; Chang, Sang-Ah; Chu, James W.; Ciaraldi, Theodore P.; Lamendola, Cindy; McLaughlin, Tracey; Reaven, Gerald M.; Reaven, Peter D.

LOCATION: Department of Medicine, Stanford University School of Medicine,
Stanford, CA, USA

JOURNAL: Am. J. Physiol. (American Journal of Physiology) DATE: 2006

VOLUME: 290 NUMBER: 1, Pt. 2 PAGES: R139-R144 CODEN: AJPHAP

ISSN:

0002-9513 LANGUAGE: English PUBLISHER: American Physiological Society

SECTION:

CA201010 Pharmacology

CA202XXX Mammalian Hormones

IDENTIFIERS: thiazolidinedione insulin resistance wt loss adiponectin

multimer rosiglitazone

DESCRIPTORS:

Cytokines...

adiponectin, including multimeric complexes; improvements in insulin

resistance with wt. loss, in contrast to rosiglitazone, are not assocd.

with changes in plasma adiponectin or adiponectin multimeri Human... Obesity...

improvements in insulin resistance with wt. loss, in contrast to rosiglitazone, are not assocd. with changes in plasma adiponectin or

adiponectin multimeric complexes

Body weight...

loss; improvements in insulin resistance with wt. loss, in contrast to

rosiglitazone, are not assocd. with changes in plasma adiponectin or

adiponectin multimeric complexes

Diet...

restricted; improvements in insulin resistance with wt. loss, in

contrast to rosiglitazone, are not assocd. with changes in plasma adiponectin or adiponectin multimeric complexes

CAS REGISTRY NUMBERS:

50-99-7 biological studies, blood; improvements in insulin resistance with

wt. loss, in contrast to rosiglitazone, are not assocd. with changes in

plasma adiponectin or adiponectin multimeric complexes

9004-10-8 biological studies, improvements in insulin resistance with wt.

loss, in contrast to rosiglitazone, are not assocd. with changes in

plasma adiponectin or adiponectin multimeric complexes

122320-73-4 improvements in insulin resistance with wt. loss, in contrast

to rosiglitazone, are not assocd. with changes in plasma adiponectin or

adiponectin multimeric complexes

3/7/107 (Item 8 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

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143432670 CA: 143(24)432670e PATENT

Materials and methods for modulating metabolism

INVENTOR(AUTHOR): Chan, Bill Piu; Wong, Gary Kwan Po; Xu, Jinxian; Chi, Francis

LOCATION: Peop. Rep. China,

PATENT: U.S. Pat. Appl. Publ. ; US 20050245433 A1 DATE: 20051103

APPLICATION: US 2005118737 (20050429) *US 2004PV567899 (20040503)

*US

2004PV637618 (20041220)

PAGES: 23 pp. CODEN: USXXCO LANGUAGE: English

PATENT CLASSIFICATIONS:

CLASS: 514003000; A61K-038/28A; A61K-031/4439B; A61K-031/426B; A61K-031/13B

SECTION:

CA201010 Pharmacology

CA263XXX Pharmaceuticals

IDENTIFIERS: cysteamine compn hypercholesterolemia therapy diabetes

DESCRIPTORS:

Adipose tissue...

adipocyte, glut4 expression in; compns. and methods for treatment of

metabolic diseases

Cytokines...

adiponectin; compns. and methods for treatment of metabolic diseases

Adrenoceptor antagonists...

α -; compns. and methods for treatment of metabolic diseases

Heart,disease...
 angina pectoris; compns. and methods for treatment of metabolic diseases
Adrenoceptor antagonists...
 β -; compns. and methods for treatment of metabolic diseases
Hemorrhage...
 cerebral; compns. and methods for treatment of metabolic diseases
Fatty acids,biological studies... Hypercholesterolemia...
Anticholesteremic
agents... Hypolipemic agents... Obesity... Antiobesity agents...
Cardiovascular system,disease... Cardiovascular agents...
Hypertension...
Antihypertensives... Hyperglycemia... Arteriosclerosis...
Antiarteriosclerotics... Antianginal agents... Prophylaxis...
Ischemia...
Anti-ischemic agents... Combination chemotherapy... Fibrates...
Platelet
aggregation inhibitors... Anticoagulants... Antidiabetic agents...
Sulfonylureas... Encapsulation... Proteins... Crown ethers...
Polyoxyalkylenes,biological studies... Polysiloxanes,biological studies...
Particle size... Bacilli... Human...
 compns. and methods for treatment of metabolic diseases
Thrombosis...
 coronary arterial; compns. and methods for treatment of metabolic diseases
Artery,disease...
 coronary, thrombosis; compns. and methods for treatment of metabolic diseases
Artery,disease...
 coronary; compns. and methods for treatment of metabolic diseases
Metabolism,animal...
 disorder, glucose intolerance; compns. and methods for treatment of metabolic diseases
Transport proteins...
 glucose transporter; compns. and methods for treatment of metabolic diseases
Transport proteins...
 GLUT-4 (glucose transporter 4); compns. and methods for treatment of metabolic diseases
Liver... Muscle...
 glut4 expression in; compns. and methods for treatment of metabolic diseases
Drug delivery systems...
 granules, enteric-coated; compns. and methods for treatment of metabolic diseases
Drug delivery systems...

granules; compns. and methods for treatment of metabolic diseases
Brain,disease...
hemorrhage; compns. and methods for treatment of metabolic
diseases
Lipoproteins...
high-d.; compns. and methods for treatment of metabolic diseases
Lipids,biological studies...
hyperlipidemia; compns. and methods for treatment of metabolic
diseases
Glycerides,biological studies...
hypertriglyceridemia; compns. and methods for treatment of
metabolic
diseases
Histamine receptors...
H2, blocker; compns. and methods for treatment of metabolic
diseases
Autoimmune disease...
insulin-dependent diabetes mellitus; compns. and methods for
treatment
of metabolic diseases
Diabetes mellitus...
insulin-dependent; compns. and methods for treatment of metabolic
diseases
Artery,disease...
intermittent claudication; compns. and methods for treatment of
metabolic diseases
Arm... Leg...
ischemia in; compns. and methods for treatment of metabolic
diseases
Lipoproteins...
low-d.; compns. and methods for treatment of metabolic diseases
Albumins,biological studies...
microalbumin; compns. and methods for treatment of metabolic
diseases
Diabetes mellitus...
non-insulin-dependent; compns. and methods for treatment of
metabolic
diseases
Antidiabetic agents...
oral; compns. and methods for treatment of metabolic diseases
Drug interactions...
pharmacodynamic; compns. and methods for treatment of metabolic
diseases
Bile acids...
resins; compns. and methods for treatment of metabolic diseases
Drug delivery systems...
solids; compns. and methods for treatment of metabolic diseases
Brain,disease...
stroke; compns. and methods for treatment of metabolic diseases
Zeolites(synthetic),biological studies...
Zeolites (synthetic); compns. and methods for treatment of
metabolic

diseases

CAS REGISTRY NUMBERS:

50-99-7 biological studies, blood, dysglycemia; compns. and methods for

treatment of metabolic diseases

69-93-2 biological studies, blood, hyperuricemia; compns. and methods for

treatment of metabolic diseases

59-67-6 9005-25-8 biological studies, compns. and methods for treatment

of metabolic diseases

9004-10-8 biological studies, hyperinsulinemia; compns. and methods for

treatment of metabolic diseases

12619-70-4D branched, compns. and methods for treatment of metabolic diseases

60-23-1 61912-98-9 59112-80-0 156-57-0 75330-75-5 81093-37-0

79902-63-9 93957-54-1 134523-00-5 56211-40-6 50-78-2 56-03-1

2295-31-0 673-06-3 10238-21-8 29094-61-9 93479-97-1 64-77-7

94-20-2 135062-02-1 105816-04-4 56180-94-0 72432-03-2

122320-73-4

111025-46-8 11041-12-6 50925-79-6 182815-43-6 95522-45-5

52757-95-6 12619-70-4 9030-09-5 84337-62-2 107745-73-3

161973-57-5

79647-56-6 868350-96-3 657-24-9 compns. and methods for treatment of

metabolic diseases

9004-34-6D 9004-54-0D 9005-25-8D derivs., compns. and methods for treatment of metabolic diseases

9015-82-1 9001-42-7 inhibitor; compns. and methods for treatment of metabolic diseases

3/7/108 (Item 9 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

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142407211 CA: 142(22)407211c PATENT

Method of separating and assaying adiponectin multimer

INVENTOR(AUTHOR): Ebinuma, Hiroyuki; Yago, Hirokazu; Akimoto, Yuka; Miyazaki, Osamu; Kadowaki, Takashi; Yamauchi, Toshimasa; Hara, Kazuo

LOCATION: Japan,

ASSIGNEE: Daiichi Pure Chemicals Co., Ltd.; Toudai Tlo, Ltd.

PATENT: PCT International ; WO 200538457 A1 DATE: 20050428

APPLICATION: WO 2004JP15260 (20041015) *JP 2003354930 (20031015)

PAGES: 40 pp. CODEN: PIXXD2 LANGUAGE: Japanese

PATENT CLASSIFICATIONS:

CLASS: G01N-033/53A; G01N-027/447B

DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BW; BY;

BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; EG; ES; FI;

GB; GD;

GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK;
LR; LS;
LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NA; NI; NO; NZ; OM; PG;
PH; PL;
PT; RO; RU; SC; SD; SE; SG; SK; SL; SY; TJ; TM; TN; TR; TT; TZ; UA;
UG; US;
UZ; VC; VN; YU; ZA; ZM; ZW DESIGNATED REGIONAL: BW; GH; GM; KE; LS;
MW; MZ
; NA; SD; SL; SZ; TZ; UG; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM;
AT;
BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; HU; IE; IT; LU;
MC; NL;
PL; PT; RO; SE; SI; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW;
ML; MR;
NE; SN; TD; TG

SECTION:

CA209016 Biochemical Methods

CA214XXX Mammalian Pathological Biochemistry

IDENTIFIERS: adiponectin ELISA proteinase sepg multimer human blood
disease diagnosis

DESCRIPTORS:

Cytokines...

adiponectin, including various multimers from human blood; method
of

sepg. and assaying adiponectin multimer

Immunoassay...

enzyme-linked immunosorbent assay; method of sepg. and assaying
adiponectin multimer

Disease, animal...

metabolic syndrome X; method of sepg. and assaying adiponectin
multimer

Disulfide group... Immunoassay... Human... Blood...

Albumins, reactions...

Kidney, disease... Liver, disease... Arteriosclerosis... Obesity... Gel
permeation chromatography... Gel electrophoresis...

Digestion, chemical...

Sample preparation...

method of sepg. and assaying adiponectin multimer

Diabetes mellitus...

non-insulin-dependent; method of sepg. and assaying adiponectin
multimer

Antibodies and Immunoglobulins...

to albumin, to adiponectin; method of sepg. and assaying
adiponectin

multimer

CAS REGISTRY NUMBERS:

9001-92-7 9003-05-8 9014-01-1 66676-43-5 209864-06-2 305344-27-8
method of sepg. and assaying adiponectin multimer

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142322694 CA: 142(17)322694n PATENT

Adiponectin secretion enhancers containing plant extracts and/or their

microbial conversion products, and their use in antiarteriosclerotics,

antiobesity agents, antidiabetics, food additives, functional foods, and

feed additives

INVENTOR(AUTHOR): Akihisa, Toshihiro; Kobayashi, Masaki; Higashio, Chie;

Takahashi, Akira

LOCATION: Japan,

ASSIGNEE: Enkaku Iryou-Laboratories Co., Ltd.

PATENT: Japan Kokai Tokkyo Koho ; JP 200568132 A2 DATE: 20050317

APPLICATION: JP 2004143282 (20040513) *JP 2003287984 (20030806)

PAGES: 21 pp. CODEN: JKXXAF LANGUAGE: Japanese

PATENT CLASSIFICATIONS:

CLASS: A61K-035/78A; A23K-001/16B; A23L-001/30B; A61K-031/07B; A61K-031/575B; A61K-031/704B; A61P-003/04B; A61P-003/10B;

A61P-009/10B;

C12Q-001/68B

SECTION:

CA263004 Pharmaceuticals

CA201XXX Pharmacology

CA211XXX Plant Biochemistry

CA217XXX Food and Feed Chemistry

CA218XXX Animal Nutrition

IDENTIFIERS: adiponectin secretion enhancer plant ext

antiarteriosclerotic, rice Momordica chrysanthemum adiponectin

secretion enhancer, rye Betula Alpinia adiponectin secretion enhancer,

shimeji ergosterol adiponectin secretion enhancer antiobesity,

antidiabetic adiponectin secretion enhancer plant ext, food feed

additive adiponectin secretion enhancer

DESCRIPTORS:

Sterols... Triterpenes... Antiarteriosclerotics... Antiobesity agents...

Antidiabetic agents... Food additives... Health food... Feed additives...

Blood serum... Drug bioavailability... Lyophyllum aggregatum...

Chrysanthemum... Secale cereale... Betula platyphylla japonica...

Alpinia

zerumbet... Hypsizygus marmoreus...

adiponectin secretion enhancers contg. plant exts. and/or their

microbial conversion products for antiarteriosclerotics,

antiobesity

agents, antidiabetics, food additives, functional foods, and feed

a

Cytokines...

adiponectin, secretion enhancers; adiponectin secretion enhancers

contg. plant exts. and/or their microbial conversion products for
 antiarteriosclerotics, antiobesity agents, antidiabetics, food
 addit
Oryza sativa...
 bran; adiponectin secretion enhancers contg. plant exts. and/or
 their
 microbial conversion products for antiarteriosclerotics,
 antiobesity
 agents, antidiabetics, food additives, functional foods, and
 Gene, animal...
 expression; adiponectin secretion enhancers contg. plant exts.
 and/or
 their microbial conversion products for antiarteriosclerotics,
 antiobesity agents, antidiabetics, food additives, functional
 foods
Momordica grosvenori...
 fruit; adiponectin secretion enhancers contg. plant exts. and/or
 their
 microbial conversion products for antiarteriosclerotics,
 antiobesity
 agents, antidiabetics, food additives, functional foods, and
 Peroxisome proliferator-activated receptors...
 γ , gene expression enhancement; adiponectin secretion enhancers
 contg. plant exts. and/or their microbial conversion products for
 antiarteriosclerotics, antiobesity agents, antidiabetics, food a
 Bran...
 rice; adiponectin secretion enhancers contg. plant exts. and/or
 their
 microbial conversion products for antiarteriosclerotics,
 antiobesity
 agents, antidiabetics, food additives, functional foods, and
 Arteriosclerosis... Obesity... Diabetes mellitus...
 therapeutic agents; adiponectin secretion enhancers contg. plant
 exts.
 and/or their microbial conversion products for
 antiarteriosclerotics,
 antiobesity agents, antidiabetics, food additives, function
 CAS REGISTRY NUMBERS:
 848168-94-5 848168-95-6 848168-96-7 848168-97-8 848168-98-9
 848168-99-0 848169-00-6 848169-01-7 848169-02-8 848169-03-9
 adiponectin secretion enhancers contg. plant exts. and/or their
 microbial conversion products, and their use in
 antiarteriosclerotics,
 antiobesity agents, antidiabetics, food additives, functional
 foods,
 and feed additives
 469-38-5 57576-29-1 88901-36-4 57-87-4 35176-46-6 21238-33-5
 106774-76-9 409349-92-4 adiponectin secretion enhancers contg.
 plant
 exts. and/or their microbial conversion products for
 antiarteriosclerotics, antiobesity agents, antidiabetics, food
 additives, functional foods, and feed additives

3/7/110 (Item 11 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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142273179 CA: 142(15)273179n JOURNAL
Extrapaneatic effects of glimepiride - focusing on
antiarteriosclerotic
action
AUTHOR(S): Takayama, Hideichi
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JOURNAL: BIO Clin. (BIO Clinica) DATE: 2004 VOLUME: 19 NUMBER: 13
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PUBLISHER: Hokuryukan
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IDENTIFIERS: review glimepiride antiarteriosclerotic
arteriosclerosis
insulin resistance platelet
DESCRIPTORS:
Cytokines...
adiponectin; extrapaneatic effects of glimepiride as
antiarteriosclerotic agent
Endothelium...
coronary arterial; extrapaneatic effects of glimepiride as
antiarteriosclerotic agent
Artery...
coronary, endothelium; extrapaneatic effects of glimepiride as
antiarteriosclerotic agent
Antiarteriosclerotics... Arteriosclerosis... Platelet aggregation
inhibitors...
extrapaneatic effects of glimepiride as antiarteriosclerotic
agent
Lipids,biological studies...
metab.; extrapaneatic effects of glimepiride as
antiarteriosclerotic
agent
CAS REGISTRY NUMBERS:
10102-43-9 biological studies, extrapaneatic effects of
glimepiride as
antiarteriosclerotic agent
9004-10-8 biological studies, resistance; extrapaneatic effects of
glimepiride as antiarteriosclerotic agent
93479-97-1 extrapaneatic effects of glimepiride as
antiarteriosclerotic
agent
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S2	464	S1 AND (MULTIMER OR AGGREGAT?)

S3 110 RD S2 (unique items)
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---Logging off of Dialog---

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